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VOLUME I

DISEASES OF THE SPINAL CORD AND NERVES

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OF

DISEASES OF THE NERVOUS SYSTEM

BY

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PREFACE TO VOL. II.

THE delay in the appearance of the second edition of this volume has been due to the time needed for its thorough revision, and the incorporation of the chief results of the investigations of the last five years. Numerous additions have been made, and many sections have been re-written, in the endeavour to present, as far as possible, the current knowledge of the subjects considered. In spite of some condensation these additions involve an increase of about a hundred pages.

I am much indebted to M. Richer for permitting me to reproduce some of his very effective illustrations of the phenomena of the attacks of "Hysteria major," and trust that the figures will serve to make his work, clinical and artistic, more widely known in this country.

For the index, and also for most useful help in the process of revision and in the correction of the proofs, I have to thank my friend and colleague, Dr. James Taylor.

W. R. GOWERS.

QUEEN ANNE STREET, LONDON.

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DISEASES OF THE NERVOUS SYSTEM.

PART IV.

DISEASES OF THE BRAIN.

INTRODUCTION.

THE STRUCTURE AND FUNCTIONS OF THE BRAIN.

The knowledge we possess of the cerebral structure and functions is derived from various sources. Simple dissection of the brain reveals only the coarser outlines of its anatomy, and is apt to mislead the investigator who endeavours, by its aid alone, to unravel the complex connection of the several parts. By the aid of the microscope the structure of isolated portions can be ascertained, but the difficulty and even impossibility of tracing the course of nerve-fibres, where thousands interlace, limits narrowly the results to be obtained from simple microscopical examination. But the differentiation effected by processes of degeneration on the one hand, and of development on the other, has enabled many facts to be ascertained which are of the highest importance, and which must otherwise have remained unknown. The close correspondence between the results obtained by these two methods, the study of development and of degeneration, not only shows their high value, but also justifies confidence in the indications afforded by either, where the two cannot be combined. The credit of discovering the fact and significance of secondary degeneration belongs to Türck, and Flechsig first showed the value of the structural indications of development, by investigations which have placed many points in the anatomy of the brain on a footing far firmer than any other method could supply. These investigations have been ably extended by Bechterew.

The functions of the brain have been ascertained by three methods. First, the structural arrangement of its parts affords important suggestions as to their function—suggestions which are valuable in proportion to the simplicity of the structure, and the distinctness of its connections. For instance, a group of nerve-cells gives origin to the fibres of a cranial nerve, and from this it is inferred that the function of these cells is to excite the nerve-fibres if the nerve is motor, or to receive the impulses which traverse them if the nerve is sensory. Another source of knowledge is the result of experiments upon animals, in which portions of the brain are removed, or destroyed, or stimulated, and the effects are observed. The results obtained in this manner by Hitzig, Ferrier, Munk, and others, are of very great importance; so far as the human brain is concerned they are suggestive, often highly suggestive, but they are not demonstrative, and the conclusions thus reached cannot be accepted as certainly true of man except in so far as they receive confirmation from the third source of knowledge, the experiments wrought by processes of disease, by accidental violence, or surgical procedure. It is to these that we have to look for exact knowledge and certain proof of the functions of the several parts of the brain of man. Unfortunately, the conditions of disease and injury are usually very complex, and their manifestations are correspondingly involved and difficult to interpret. Hence our knowledge grows but slowly, in spite of the enormous amount of careful observation which, at the present day, is directed to this subject in all parts of the world.

The following account of the structure and functions of the brain does not profess to be complete, even in outline. Its object is to remind the student of the facts he most needs to know in order to comprehend the symptoms and pathology of disease. Unfortunately, our knowledge is still imperfect, even of the fundamental facts. Many questions of great importance are still unanswered, while to others widely different answers have been given by investigators of equal competence. The statements here made on such points are the result not only of a comparison of the work of others, but also, in many instances, of an independent examination of the point at issue. The functions of the brain will be considered very briefly, since many of them must be discussed more fully in the account of the symptoms of disease.

The few points in the anatomy of the membranes which are of medical importance will be most conveniently considered in the section on their diseases.

THE CEREBRAL CORTEX.

The anatomy of the convolutions of the brain has become of great practical importance, on account of the definite localisation of function found to obtain in certain parts. For a full account of the convolutions the reader is referred to works on Anatomy, but the most important facts may be briefly enumerated. The *longitudinal fissure* separates the two hemispheres. In each half of the brain three fissures are of leading importance (see Fig. 1), the fissures of Sylvius and of Rolando on the outer surface, and the parieto-occipital fissure, the extremity only of which appears on the convexity. The *Sylvian fissure*, at the base, separates the prominent temporal lobe from the orbital surface of the frontal lobe, and, on the outer surface, divides into a very short anterior limb and longer posterior limb; sometimes there are two short limbs, one anterior and the other vertical (see Figs. 1 and 3). The posterior limb separates the temporal lobe below from the parietal lobe above. Just above the bifurcation of the fissure of Sylvius, but not joining it, is the lower extremity of the *fissure of Rolando*, or *central sulcus*, which passes upwards and backwards to the

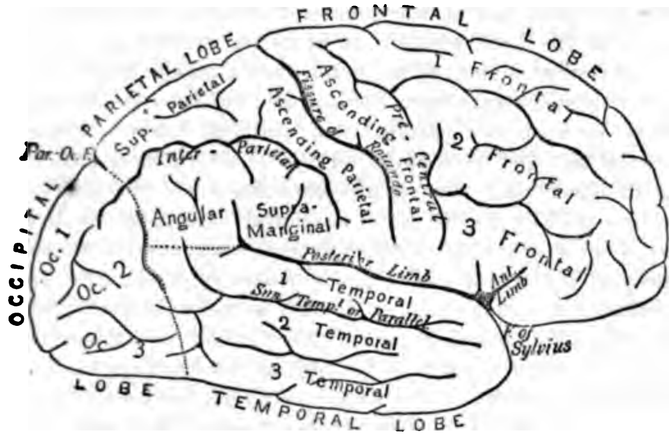

FIG. 1.—Diagram of the convolutions and fissures on the outer surface of the right hemisphere. The fissures are indicated by italics.

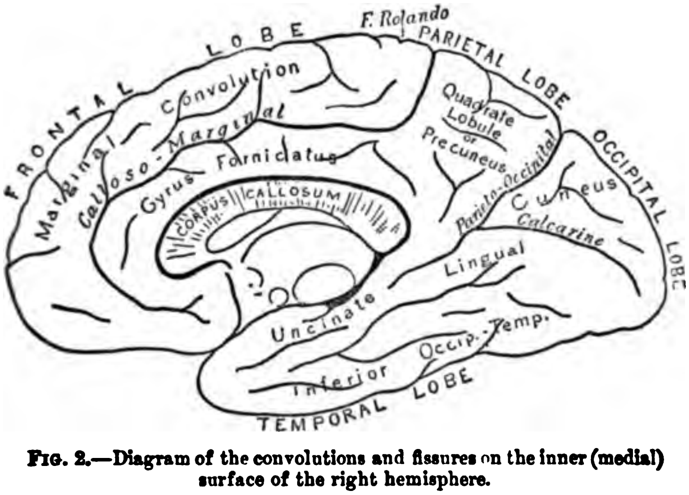

FIG. 2.—Diagram of the convolutions and fissures on the inner (medial) surface of the right hemisphere.

longitudinal fissure, and separates the frontal from the parietal lobe. It presents in its course two bends, the superior and inferior genu, of which the latter, roughly speaking opposite the upper part of the middle frontal convolution, is an important landmark. The *parieto-occipital fissure* begins at the longitudinal fissure, about midway between the upper end of the fissure of Rolando and the posterior extremity of the hemisphere. It is better marked on the medial than on the outer surface of the hemisphere; on the latter it extends only for a short distance from the longitudinal fissure. It marks the boundary between the parietal and occipital lobes. These three fissures are the chief landmarks on the outer surface, and are our guides in identifying the various convolutions. On the inner or medial aspect (Fig. 2) the parieto-occipital fissure, at its lower extremity, joins another sulcus which extends backwards to the posterior extremity of the brain, the *calcarine fissure*, and the two bound a wedge-shaped area, the *cuneus*. In the anterior half of this inner surface, the *calloso-marginal fissure* runs parallel with the corpus callosum, midway between it and the edge of the hemisphere, to which the posterior extremity of this fissure turns up, behind the upper end of the fissure of Rolando. Between this upcurved end and the cuneus is an area termed the *pre-cuneus*, or, from its shape, the *quadrate lobule*.

The fissure of Rolando runs between two convolutions which have the same direction as the fissure. These are the *ascending frontal* and *ascending parietal* convolutions. These two gyri are of great importance, because they contain the chief so-called "motor" centres. They are sometimes termed the "central convolutions," "anterior" and "posterior." They unite below the lower end of the fissure of Rolando, and the region that occupies the bifurcation of the fissure of Sylvius is often termed the "operculum." The ascending frontal is bounded by the *pre-central sulcus*, usually interrupted by the origin of one or two of the remaining frontal convolutions, *upper*, *middle*, and *lower;* or *first*, *second*, and *third*. The highest is at the margin of the longitudinal fissure; the lowest curves round the anterior limb of the fissure of Sylvius. The upper part of the ascending parietal convolution blends with the upper part of the parietal lobe in the *superior parietal lobule*, also called the parietal lobule; this also forms part of the motor region, and so does the medial aspect of these two central convolutions, the *paracentral lobule*, it is termed, in front of the upcurved end of the calloso-marginal fissure. The lower part of the parietal lobe, behind the ascending parietal convolution, is termed the *inferior parietal lobule*, and is separated from the superior lobule by the *interparietal*, also called the *intraparietal*, *fissure*.

The lowest parietal convolution, which bounds, above, the posterior limb of the fissure of Sylvius, is termed the *supra-marginal convolution*. In some brains this constitutes almost the whole of the inferior parietal lobule. By some authorities (Turner, &c.) the supra-marginal gyrus is regarded as ceasing above the extremity of the upturned end of the fissure of Sylvius; by others it is regarded as passing round this, so that the gyrus immediately behind the upturned portion of the fissure is included in the name. Behind this upturned fissure is the *angular gyrus*, which extends back to the occipital lobe, upwards to the intra-parietal sulcus, and downwards is continuous with the first and second temporal convolutions. Its posterior limit is somewhat arbitrary, corresponding to a line drawn across from the parieto-occipital fissure. Its anterior limit varies according to the area assigned to the supra-marginal convolution.*

The convolutions passing from the parietal to the occipital lobes on the outer surface are called "annectant;" the highest of these is at the outer extremity of the short parieto-occipital fissure.

In the *occipital lobe* three short convolutions may often be distinguished,—*upper*, *middle*, and *lower*. The upper one is continuous with the superior parietal lobule by the first annectant gyrus. These occipital convolutions are often not distinctly separate. In the *temporal lobe* two or three antero-posterior convolutions can be distinguished on the outer surface. Of these the *upper*, or *first*, forms the inferior boundary to the fissure of Sylvius, and is continuous behind with the supra-marginal or the angular convolution, or with both, according to the sense in which these words are used. The fissure below this convolution is termed the *parallel fissure*, because it is parallel with the fissure of Sylvius. The lower convolution or convolutions (if two can be distinguished) are continuous with the occipital lobe.

On the medial surface of the temporal lobe (Fig. 2) two or three similar convolutions are visible; and of these the upper one is the *uncinate* gyrus, so called because its anterior extremity is shaped like a hook. Beneath the calcarine fissure is the ***lingual gyrus***. In the rest of the medial surface the ***gyrus fornicatus*** lies next to the corpus callosum, but only constitutes a well-marked convolution in the anterior half of the brain, where the calloso-marginal fissure separates it from the ***marginal convolution***, which is, for the most part, the medial aspect of the highest frontal convolution. The other divisions of the medial surface have been already enumerated. Within the fissure of Sylvius lie the small convolutions of the ***island of Reil***, or ***insula*** (Fig. 3), four or five in number (I to V, Fig. 3), which spread out like a fan. Behind the insula, but still entirely within the fissure of Sylvius, two or three convolutions connect the first temporal with the parietal lobe. They have been termed the ***retro-insular*** or ***temporo-parietal convolutions*** (R I, Fig. 3). Neither these convolutions nor those of the insula are visible until the lips of the Sylvian fissure are separated. Hence they often escape examination, and this is probably one reason why little is known of the effect of their isolated disease. It will be observed that the boundary between the frontal and parietal lobes also separates the two "ascending" convolutions, which have been found to have functions at once similar, important, and unlike those of most other parts of the brain. Hence it is often convenient to distinguish that part of the frontal lobe which lies in front of the ascending convolution as the "pre-frontal lobe."

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* The importance attached to the angular convolution renders it desirable to point out the different senses in which the word is used. The term was taken from the "*pli courbe*" of Gratiolet, applied by him to the convolution in the brain of the ape which winds round the top of the fissure of Sylvius, and, as a simple gyrus, is limited behind by the parieto-occipital fissure, which extends, in the ape's brain, far on the outer surface of the hemisphere. But in applying the term to the more complex brain of man, Gratiolet restricted it to the posterior part of the region between the extremity of the fissure of Sylvius and the occipital lobe. The term is now used in four senses. (1) The whole region from the end of the fissure of Sylvius to the occipital lobe. (2) The posterior two thirds of this region, a small sulcus, parallel with the upturned fissure of Sylvius, being taken as the anterior limit of the angular and posterior limit of the supra-marginal, which is thus regarded as passing round the fissure of Sylvius. In this sense the parallel fissure passes into the angular gyrus. (3) The posterior third of this region; the anterior limit being the parallel fissure, to which the supra-marginal will then extend. (4) The term is sometimes used (and the corresponding "*pli courbe*" by French writers) to include not only the whole of this region, but also that part of the supra-marginal gyrus which lies in front of the extremity of the fissure of Sylvius. This makes it correspond to the whole region into which the *pli courbe* of the ape has developed in the human brain; but it is inconsistent with the application of the word to the human brain by Gratiolet himself and most other authorities.

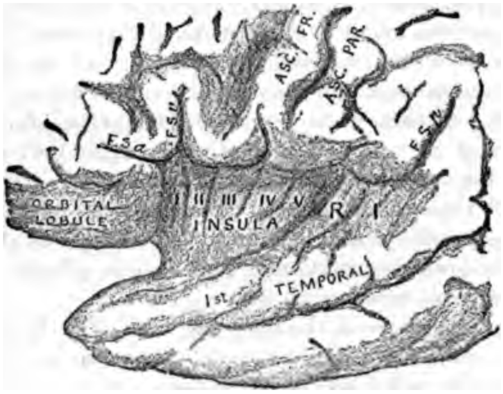

Fig. 3.—Convolutions within the fissure of Sylvius. F S *a* its anterior and F S *v* its vertical, and F S *p* the extremity of its posterior limb. I—V, the short gyri of the insula; R I, retro-insular convolutions connecting the temporal and parietal lobes.

RELATION OF THE CORTEX TO THE SKULL.—The correspondence between certain parts of the surface of the brain and of the skull has been carefully investigated, especially by Broca. Kefftler, Turner, Championnière, Féré, Hare, Poirier, Le Fort, Reid, Horsley, and Cunningham, and rules have been formulated by which the position of the various fissures and convolutions may be determined. The general relation of the brain to the bones of the skull is shown in Fig. 4. It presents some degree of variation in different individuals,* but this is seldom enough to have been found important. Beneath the frontal bone lies nearly all the lowest frontal, five sixths of the middle, and about three quarters of the upper frontal convolutions. The temporal bone covers the temporal lobe except its posterior fifth and anterior extremity. The major part of the occipital lobe lies under the occipital bone. The rest of the cerebral cortex lies beneath the parietal bone.

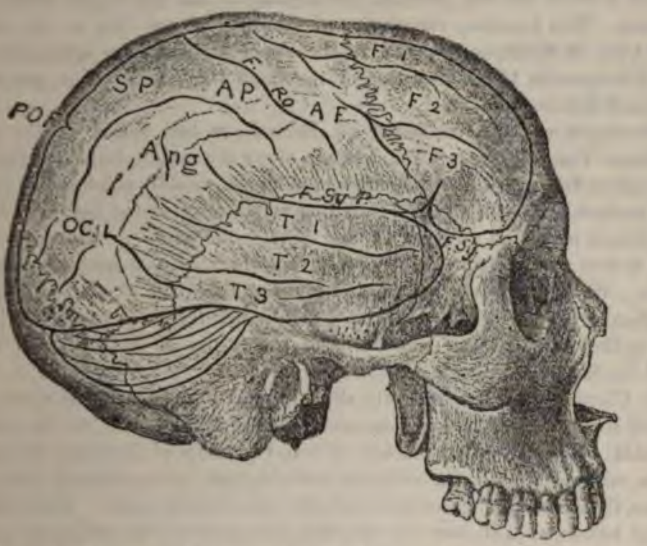

FIG. 4.—Diagram of the relation of the convolutions to the skull. F 1, 2, and 3, upper, middle, and lower frontal convolutions; A F, A P, ascending frontal and parietal; S P, sup. parietal lobule; Ang, angular gyrus; Oc L, occipital lobe; T 1, 2, 3, the temporal convolutions; P O F, parieto-occipital fissure; F Sy and F Sy P, fissure of Sylvius and its posterior limb.†

Horsley's Rules.—My colleague, Professor Victor Horsley, who has had so much practical experience in the surgery of the brain, employs a method of finding the position of the important centres of the brain, of which he has published an account in the 'International Journal of Medical Science' for April, 1887. From this the following abstract is taken:

The chief landmarks on the surface of the skull are (1) the parietal eminence and (2) the curved temporal ridge, which is really double; a lower one which is best marked and limits the attachment of the temporal muscle, and an upper one to which the temporal fascia is attached. The lower one can be best found by making the patient contract the muscle firmly; the upper one marks the sudden change in the slope of the skull to the vertical direction. (3) The coronal suture, which can generally be felt just above the place where the temporal ridge crosses it (this crossing is termed by Broca the stephanion). If the upper part cannot be felt, its position can be ascertained by finding its junction with the sagittal suture.

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* See Anderson and Makins, 'Journ. Anat. and Phys.,' 1889.

† In this figure the highest part of the parieto-occipital fissure should be represented as lying at or a little above the apex of the lambdoidal suture.

The parieto-squamosal suture lies beneath the temporal muscle: the highest point of its curve is in a vertical line passing just in front of the articulation of the lower jaw, and it is there two thirds of the distance from the zygoma to the temporal ridge.

The short junction of the anterior inferior corner of the parietal bone with the wing of the sphenoid (corresponding nearly to the division of the Sylvian fissure) is about halfway between the stephanion and the upper border of the zygoma. This junction, the pterion of Broca, is shaped like an H, the horizontal bar of which corresponds to the upper border of the sphenoidal wing, and it is opposite the hinder end of the same that the division point of the fissure of Sylvius lies.

The upper end of the fissure of Rolando is best found by the plan of Professor Thane. Measure the distance along the middle line from the root of the nose to the occipital protuberance, and find the middle point of this distance; half an inch behind this point is the upper extremity of the fissure of Rolando. The fissure makes an angle with the middle line of 67°. Professor Horsley used at first a long strip of soft metal, from which projects another piece at this angle. When this strip is laid along the middle line of the head, with the junction of the two parts over the spot mentioned, the arm corresponds in direction to that of the upper two thirds of the fissure of Rolando. In its lower third the fissure is rather more vertical.

Dr. Claud Wilson subsequently slightly modified Mr. Horsley's instrument, so that it consisted of two pieces, one at a fixed angle of 67° to the other, but movable. Further, it was marked off into two series of divisions, the posterior series, corresponding to the occipital protuberance, being lettered with capital letters, the anterior being lettered with the same letters small. With one of the capital letters, *e. g.* B, over the occipital protuberance, the corresponding small letter marked the situation in the particular case of the upper end of the fissure of Rolando, and on running the movable limb along the horizontal one the former corresponded in situation and direction with that sulcus.

Mr. Horsley has introduced more recently an important modification of this instrument. From measurements made on a series of specimens prepared by Professor Cunningham of Dublin, he found that the direction of the fissure of Rolando had apparently a definite relation to the relative breadth of the head. Thus in a dolicho-cephalic individual the angle formed by the fissure of Rolando with the longitudinal fissure was acute, while in the brachy-cephalic, on the other hand, it more and more nearly became a right angle. Such variations must of course be provided for if accuracy is to be attained in surgical procedure, and Mr. Horsley now uses an instrument in which the limb, marking out the fissure of Rolando, can be placed at a varying angle and fixed goniometrically to the limb which corresponds to the longitudinal fissure. The variation of this angle depends upon the character of the skull, whether dolicho-cephalic or brachy-cephalic, and a measure of this is obtained by taking the cephalic index in the manner which has been made familiar by anthropometric observations.

The anterior limb of the fissure of Sylvius runs upwards from the parieto-sphenoidal junction above mentioned. The posterior limb passes backwards and upwards just above the ascending part of the parieto-temporal suture, and from the highest part of this it curves upwards towards the centre of the parietal eminence. This is only true for the adult.

The lower half of the pre-central sulcus is parallel to and behind the coronal suture. The interparietal sulcus lies, in its ascending part, midway between the line of the fissure of Rolando and the parietal eminence, and then passes backwards midway between the latter and the middle line of the skull.

The ascending frontal convolution begins below beneath the anterior inferior angle of the parietal bone, in front of the prolonged line of the fissure of Rolando, between this and the prolonged line of the lower part of the pre-central sulcus; in front of the latter will be the root of the lower frontal; behind the ascending frontal, the root of the ascending parietal.*

Reid's Rules.—The most important of Reid's rules are the following: some additions are enclosed in brackets. As a base is taken a line running from the inferior margin of the orbit (Fig. 5) through the middle of the auditory meatus. The posterior limit of the fissure of Sylvius lies beneath the hinder three fifths of a line drawn from the external angular process of the frontal bone to a point three quarters of an inch below the most prominent part of the parietal bone (this may be termed the Sylvian line). The anterior limb of the fissure ascends from this line above the middle of the zygoma. The temporal ridge corresponds nearly to the fissure between the middle and lowest frontal convolutions. If the Sylvian line is prolonged to the sagittal suture, it will give the position of the parieto-occipital fissure (and the occipital lobe, half-vision centre, lies between this and the base line). A perpendicular from the base line,

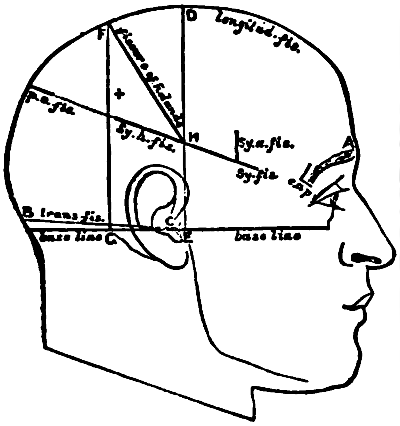

FIG. 5.—Guiding lines of Reid.

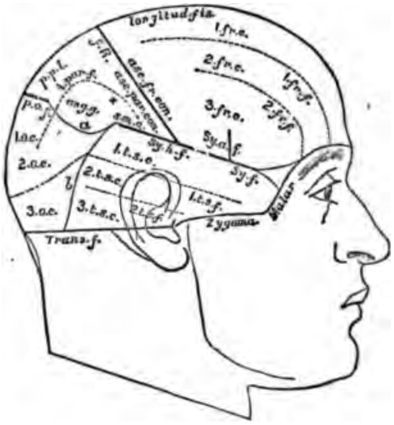

FIG. 6.—Relations of the fissures and convolutions to the guiding lines.†

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* On this subject Professor Horsley has recently contributed a chapter in Professor Cunningham's memoir on "The Surface Anatomy of the Primate Cerebrum" (Cunningham Memoir VII, Royal Irish Acad., 1892). To this reference may be made for further details.

† I am indebted to Professor Reid for permission to reproduce these figures.

at the depression in front of the auditory meatus, cuts the Sylvian line where the fissure of Rolando, if prolonged, would join the Sylvian fissure, and the position of the fissure of Rolando is under a line drawn from this point to the place at which the sagittal suture is cut by a perpendicular drawn to the base line drawn from the posterior border of the mastoid process. The central convolutions occupy about an inch on each side of the Rolandic line. The position of the centres for the leg, arm, and face can thus readily be ascertained, since they lie on each side of the fissure of Rolando. The angular gyrus lies immediately behind the most prominent part of the parietal eminence. The first temporal convolution lies below the Sylvian line, over the ear and mastoid process.

STRUCTURE OF THE CORTEX.—The nerve-fibres of the white substance radiate into the cortex, passing between the nerve-cells, and in places separating these into vertical groups. The cells are of various forms and sizes, certain of which predominate at different depths from the surface, thus permitting a distinction into layers. Each layer contains many kinds of cells, but those which are in greatest number give to the layer its distinctive character. Considerable variations exist in different parts of the brain in the number, characters, and relative thickness of the layers, and their precise division has been the subject of much discussion.* There is especially a marked difference between the characters of the cortex in the central convolutions (ascending frontal, ascending parietal, and paracentral lobule) and in the rest of the outer surface of the hemisphere. It may be said generally that the three layers next the surface present nearly the same characters over the greater part of the cortex, varying, however, in thickness. The deepest layer, next the white substance, is also very uniform in its character; the chief variations are in the elements which intervene between the three superficial layers and the deepest layer.

In the central convolutions (as the ascending frontal) the layers present the cell-forms shown in the first column in Fig. 7. Above, next the surface, is a narrow *superficial* layer containing few cells, and those of doubtful nature, chiefly small corpuscles, more or less rounded. Similar corpuscles are also scattered through all the layers. This superficial stratum contains also many delicate nerve-fibres (Remak, Lockhart Clarke, Exner). Beneath this is a layer of *small pyramidal cells* densely massed, with a few nuclear elements. The cells have the apex of the pyramid turned towards the surface. The next layer is composed of *large pyramidal* cells, less densely massed, and partially separated into columns by the bundles of nerve-fibres. The cells in the upper part of the layer are little larger than those of the second layer, but they increase in size in the deeper parts. The apex of each is turned towards the surface, and from it a long process may be traced. From the centre of the base another process extends, continuous, it is said, with the axis-cylinder of a nerve-fibre, while from the angles branching processes are given off. The small pyramidal cells occur also, in fewer number, throughout this layer, and at some parts of the brain they are more numerous in the deepest part of the layer. The next layer is characterised by nerve-cells which are irregular in shape, and often triangular. Many of them resemble the motor nerve-cells of the spinal cord, and hence it has been proposed by Bevan Lewis to call this layer the *ganglion-cell layer*. The cells vary much in size. Some are small angular elements, the size of the small pyramidal cells of the second layer; others are as large as the large pyramidal cells of the third layer; but both differ from the cells of the second and third layer in being very rarely distinctly pyramidal in form, and in being less regularly placed. Moreover in this layer some cells are met with which exceed the size of the largest pyramidal cells; some are even three times the size of the latter. These are the "giant-cells" of Betz, and were once regarded as pathological, but every gradation is seen between them and the ordinary ganglion-cells of the layer. The largest cells occur in groups of two to five, and are almost confined to the central region; while the smaller ganglion-cells of this layer are met with over the greater part of the surface of the brain. Beneath this stratum is a layer in which, with a few angular cells, there are many fusiform cells. Hence it is called the *spindle-cell layer*. It is the deepest layer of the cortex, and beneath it is the white substance of the hemisphere, among the fibres of which are many nuclei and a few angular and spindle-cells, especially near the cortex. Thus the type here is five laminated.

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* They were studied first by Baillarger, thirty years ago; subsequently by Lockhart Clarke, Meynert, and others; and, more recently, they have been investigated with much care by Betz, Bevan Lewis, Golgi, Kölliker, and Ramon y Cajal.

In the region of the brain behind the central convolutions (and also in the anterior part of the frontal lobe) the structure of the cortex more or less resembles that shown in the second section in Fig. 7, which is from the first annectant gyrus. It will be seen that the first three layers, superficial, small pyramid, and large pyramid layers, closely resemble those of the other section, and so also does the deepest or spindle-celled layer. The ganglion-cell layer is rather narrow, and con-

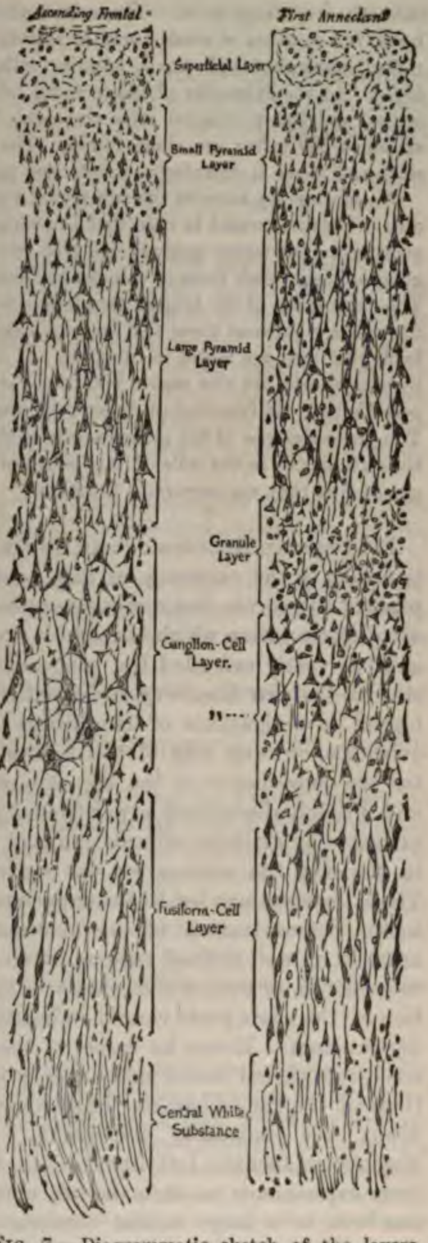

FIG. 7.—Diagrammatic sketch of the layers of the cortex cerebri. The drawings were made from sections of the ascending frontal and first annectant convolutions (the latter near the extremity of the parieto-occipital fissure). The sections were kindly furnished me, as representative of the so-called motor and sensory types, by Dr. Bevan Lewis.

tains no very large cells. Above it, and between it and the large pyramid layer, is a stratum of small round or angular granule-like elements with a few rather larger, but still small, angular cells. It has been called the *granule layer*. At the extremity of the occipital lobe this granule layer is considerably larger, and the pyramidal cells are much less developed, while in the cornu ammonis the latter are alone found. The claustrum is composed of fusiform cells, and hence is regarded as a detached part of the deepest layer.

We can merely surmise the functional significance of the several varieties of cells. It is important to note that the transition from one type of structure to another is everywhere gradual, and that (with the exception of the very large ganglion-cells) each form of cell can be found in almost all parts of the brain. The localisation of the largest ganglion-cells in the so-called motor regions, and the analogy between these and the motor cells of the cord, make it highly probable that they are motor in function. It has been conjectured that the large pyramidal cells are also motor, but from the wide extent of this formation it is possible that the function of these cells varies according to their connections. The preponderance of the granule-cells at the posterior portion of the brain, and their similarity to the cells of the posterior cornu of the spinal cord, has suggested that they are sensory in function.

Functional Regions of the Cortex.—Doubt was formerly entertained as to the existence of differentiation of function in different parts of the cortex, but recent researches have established the existence of a differentiation which has almost revolutionised cerebral physiology, and has vastly extended the range of cerebral diagnosis. The first step of the new discovery was constituted by the clinical and pathological investigations of Hughlings Jackson, which suggested the existence, on each side of the fissure of Rolando, of special centres for the movements of the leg, arm, and face. In Germany, Hitzig and Fritsch discovered experimentally that the cortex cerebri was excitable by artificial stimulation, and they succeeded in mapping out in the dog the centres for the movements of the limbs and head. These observations led to the experiments of Ferrier, which resulted in the demonstration of the existence in the cortex of the lower animals of well-defined regions, stimulation of which caused separate movements, or evidence of special sense excitation, while the destruction of the same parts caused indications of a loss of the corresponding function. Hence he came to the conclusion that these regions constitute actual motor and sensory centres. Many other investigations of the same character have since been made, of which those of Munk are especially important. The original observations of Hughlings Jackson left little doubt that the general facts, learned from experiments on animals, are true of man; and this conclusion has been to a large extent confirmed by pathological and clinical observations directed to the verification on man of the experimental results. To this verification the labours of Charcot and his coadjutors have largely contributed. But the verification has already made it probable that some differences exist between the brain of man and of other animals (even of monkeys), and that the conclusions from the latter cannot be simply transferred to the former.

Cortical Centres in the Monkey.—Before considering the indications of the position of the various centres in the cortex of the human brain, it may be well to enumerate the chief functional regions ascertained by Ferrier and others in the brain of the monkey.

Motor Centres.—*Superior parietal lobule (except the part adjacent to the fissure of Rolando):* movement of the leg and foot, flexion of the hip, extension of the knee, flexion of the ankle.

Highest and adjacent parts of the ascending frontal and parietal convolutions, close to the margin of the hemisphere, together with the root of first frontal: flexion and outward rotation of thigh, rotation inwards of leg, flexion of toes, as in scratching abdomen with foot. (Horsley and Schäfer* obtained movements of the leg also from the medial aspect of the convolutions on each side of the extremity of the fissure of Rolando. Horsley and Beevor found movement of the great toe chiefly represented in the upper extremity of the ascending frontal, and of the small toes at the extreme posterior part of the parietal lobule.)

Adjacent parts of ascending frontal and parietal, outside the last centre but still opposite the highest frontal convolution: adduction and extension of arm, pronation of hand.

Ascending frontal and base of the highest frontal: extension of elbow, movement forwards at shoulder, and synchronous movement of leg. (Horsley and Schäfer obtained movements of the arm from the medial aspect of the posterior half of the highest frontal.)

Ascending frontal, opposite upper part of middle frontal: supination of hand and flexion of forearm.

Middle three fifths of ascending parietal: movements of hand, especially clenching of fist.†

The arm area in the monkey is separated from the leg area by a small but constant sulcus which is believed to correspond to the superior pre-central sulcus in man. The lower limit is a horizontal line drawn from the extremity of the intra-parietal sulcus passing through the inferior genu of the fissure of Rolando. The same relation is found to hold in the case of the orang, and in both the orang and the monkey the thumb centre is specially localised in the region of this line.

Ascending frontal, opposite lower half of middle frontal: elevation and retraction of angle of mouth.

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* 'Proc. Roy. Soc.,' 1885.

† Horsley and Beevor ('Phil. Trans.,' 1887, 1889) found that the representation of the arm is as follows:—In the highest part of the arm region of the two central convolutions, opposite the upper frontal sulcus, the shoulder movements are represented; next, below, come the movements of the elbow behind and of the wrist in front, while in the lowest part the movements of the fingers are represented in front, and of the thumb behind. But this is only the maximum representation of the several parts, in which the special movement is produced by the weakest currents. There is scarcely any part in which movements of the elbow and wrist are not produced, or in which they are produced alone. Hence the experimenters conclude that the representation of the movement of these parts is subordinate to that of the shoulder on the one hand and of the digits on the other. In the orang, the representation of the lower limb instead of being, as in the monkey, horizontal, is vertical, the hallux being represented above and the hip below ('Phil. Trans.,' 1890).

Ascending frontal, opposite highest part of third frontal: elevation of upper lip and ala of nose.

Lowest part of ascending parietal: retraction of angle of mouth by platysma.

Lowest part of ascending frontal: movements of lips and tongue and in the anterior part, closure of the vocal cords.*

Posterior half of upper and middle frontal convolutions: lateral movement of head and eyes, with elevation of eyelids.

(*Medial aspect of top of ascending frontal, i. e.* corresponding region of the marginal convolution, movement of the trunk.—Horsley and Schäfer.)

SENSORY CENTRES.—Stimulation of the angular convolution caused a movement of the eyes to the opposite side, suggestive of a visual sensation. Extirpation of the angular gyrus caused amblyopia of the opposite eye, more or less transient, and destruction of this convolution in both hemispheres caused permanent blindness. (Munk found that hemianopia was produced by destruction of the occipital lobe, and Ferrier found that destruction of the occipital lobe and angular gyrus caused not only transient amblyopia of the opposite eye, but also permanent hemianopia. He failed to obtain any affection of vision by extirpation of the occipital lobe only, even on both sides. Nevertheless human pathology, especially the observations of Monakow and Henschen, has fully confirmed the correctness of Munk's conclusion. Horsley and Schäfer† found that destruction of one occipital lobe produced hemiopic disturbance of vision, so far confirming Munk's conclusion; but the imperfect vision which remained after destruction of both occipital lobes seemed to them to indicate the existence of visual function elsewhere, probably in the angular gyrus. The subsequent experiments of Schäfer and Sanger Brown‡ seem to confirm Munk's conclusion.)

Stimulation of the anterior part of the uncinate convolution caused movement of the nostril suggestive of a sensation of smell on the same side. Isolated extirpation of these regions is impossible, but the destruction of it, together with other parts, always caused loss of smell.

Stimulation of the superior temporo-sphenoidal convolution caused a movement suggestive of an auditory sensation on the opposite side, and destruction of this convolution caused deafness on the opposite side.

Destruction of the hippocampal region (gyrus hippocampi and hippocampus) seemed to cause partial loss of sensibility to touch and pain on the opposite side. (Horsley and Schäfer found that extensive lesions of the gyrus fornicatus had a similar effect. Munk's experiments point to the outer surface, and especially the central region, as that in which cutaneous sensibility is chiefly represented.) The experiments of Hitzig and Fritsch and Horsley's clinical observations apparently show that slight tactile sensibility and the muscular sense are represented in the so-called motor region.

CORTICAL CENTRES IN THE HUMAN BRAIN.—The evidence we possess regarding the cortical centres in the human brain is derived chiefly from the comparison of the effects of disease observed during life, with its position ascertained after death. It is probable, however, that brain surgery may indirectly increase considerably the facts at our disposal. The evidence at present available shows that there is a general correspondence between the cortical centres of man and those of the monkey. It is afforded by cases in which small lesions have caused definite loss of function, motor or sensory, or definite excitation of function, and especially by the effects of operations and the use of the facts as indications for these. The irritation symptoms are of chief significance in the case of the motor function, and are constituted by local spasm, or more commonly by convulsion beginning in or limited to, one part. But such convulsion may be caused by disease adjacent to as well as by that which is in, a cortical centre, and therefore such cases afford less precise and certain evidence than do those in which there is local destruction of tissue and local paralysis.

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* This is the centre for the vocal cord in the monkey, according to Horsley and Semon; Déjerine says it is so also in man. ('Compt. Rend.,' 1891, No. 8.)

† 'Phil. Trans.,' 1888.

‡ Ibid., 1888.

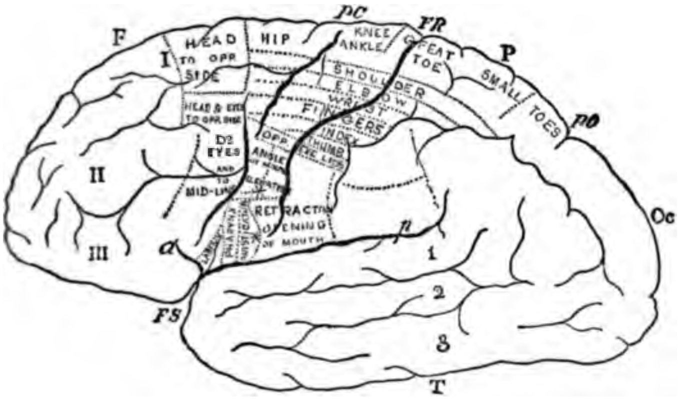

Fig. 8.—Diagram of the human brain, to which is transferred the motor centres found by Horsley in the brain of the bonnet monkey (Beevor and Horsley, 'Phil. Trans.,' vol. 181, 1890, p. 81, fig. 7). The diagram corresponds with Figs. 1 and 2. The position of the centres has been for the most part verified by Horsley in his operations. The asterisks indicate the centre for the tongue.

It is only under certain conditions, however, that even destroying lesions can be taken as evidence of functional localisation. Those conditions have been rightly insisted on by Nothnagel in his work* on this subject, a work which is a model of scientific method. It is only the lasting symptoms which can be regarded as related to the damaged region of the brain, because an acute lesion frequently causes, for a time, symptoms of much wider range than strictly correspond to the destruction. Such wide symptoms are due to pressure, secondary vascular disturbance, or irritative inhibition. Hence sufficient time must be allowed to elapse for these "indirect" effects to pass away before any inference can be drawn; that is, only such symptoms as have lasted for some weeks can be regarded as having real significance, and cases of shorter course are of small value.

Negative evidence is also important—the occurrence of lesions in certain parts without the production of certain symptoms. But another condition must be observed in the reception of this evidence. The nerve-structures are remarkably tolerant of morbid processes that develop gradually. Tumours or abscesses may form in the position of structures that are known to have a certain function, and there may be no symptoms of disturbance of that function, or such symptoms may be slight, even when the structures are apparently destroyed by the morbid processes. In these cases the nerve-elements are displaced and not destroyed; any damage they have suffered has been so slowly produced that it has not deranged their function. Hence the negative evidence afforded by such cases is of almost no value. On the other hand, these, and other lesions which cause pressure or irritation, often produce symptoms of much wider extent than corresponds to the position of the lesion or to its direct influence,—effects which are analogous in nature to the "indirect" initial consequences of an acute lesion. Hence the positive evidence afforded by irritating and compressing lesions can only be accepted with reserve. Neglect of these considerations has deprived of almost all value one of the most laborious investigations into this problem—that of Exner.*

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* 'Topische Diag. der Gehirnkrankheiten,' Berlin, 1879.

There are some functions of the cortex that elude localisation for another reason, because their loss is quickly conpensated by the other hemisphere. It appears that one-sided movements are represented in both hemispheres, and can be excited from either, in proportion as they are habitually associated on the two sides. This law, first stated by Broadbent, is of extreme importance, and we shall have to return to it many times. The above statement of it is perhaps only part of the truth, but it enables us to understand the absence of any evidence as to the position of the centres for certain movements. Before the indirect effects of a lesion have passed away, and the persistent symptoms can be admitted "into court," the loss of the movements referred to has passed away because the other hemisphere has supplied the lost function. It is so, for instance, in the case of lateral movement of the head and eyes, the movement of the muscles of mastication, and many movements of the trunk.

The evidence at present available consists of a large number of facts, ascertained by various observers, many of which have been collected and carefully compared by Nothnagel, Charcot and Pitres,† and Allen Starr.‡

Motor Centres.—There is conclusive evidence that the two ascending convolutions have the same special relation to voluntary motion as in the monkey. It is also certain that the paracentral lobule on the median aspect of these convolutions has a similar function, and that the motor region extends through part at least of the superior parietal lobule, and perhaps on to the root of the highest frontal convolution. Destructive lesions in these parts cause paralysis on the opposite side, while irritating disease causes convulsion of corresponding situation. Lesions elsewhere in the cortex cause no persistent paralysis. There is at present no direct evidence that the motor region in man extends beyond the ascending frontal, with the possible exception of the root of the first frontal.

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* 'Untersuch. u. d. Localisation der Grosshirnrinde,' Wien, 1881.

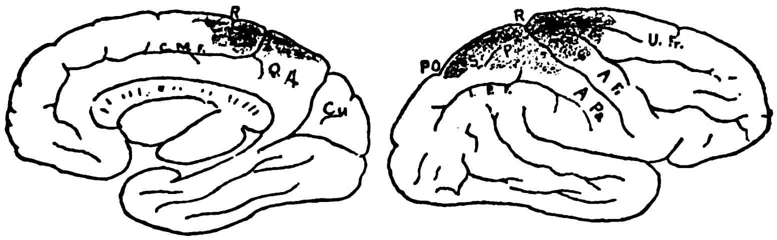
† In a valuable series of papers in the 'Revue de Méd.,' 1883.

‡ In critical and analytical collections of cases in the 'American Journal of Medical Science' and other American periodicals during the last three or four years.

It is from this motor region that the fibres arise which, as we shall see, pass down through the white substance into the anterior pyramids of the medulla, and the pyramidal tracts of the spinal cord. Through these fibres the nerve impulses pass that ultimately excite the muscles. Lesions of the cortex in this region cause a descending degeneration of these pyramidal fibres.

We can distinguish in these convolutions regions related to the leg, arm, face, and tongue, which have the same relative position as in animals.

The centre for the leg occupies the highest part of the motor region, namely, the ascending frontal and parietal convolutions, adjacent to the longitudinal fissure. We do not at present know how far back it extends in the superior parietal lobule, or how far outwards it extends on the ascending frontal.* It does not seem to extend further than the upper frontal sulcus, perhaps not so far. There may be individual variations in the area occupied by the leg centre. It certainly occupies also the paracentral lobule on the inner surface. We do not know how far the representation of separate movements corresponds to the arrangement in animals, but in many cases of convulsion beginning in the foot, the disease has been in or near the junction of the highest frontal and ascending frontal convolutions. Moreover evidence of the representation of movements of the great toe in front of the highest part of the fissure of Rolando is



FIGS. 9 and 10.—Position of the cortical centre for the leg. It is doubtful, however, whether the centre extends so far back, in the superior parietal lobule (S P), as is here represented on the outer surface.

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* See Dr. Seguin's case ('Journ. of Nerv. and Ment. Dis.,' 1887). A woman, the subject of cancer, had crural monoplegia, the result of a growth seven eighths by five eighths of an inch, subcortical, with the posterior end on a vertical level with the posterior end of the calloso-marginal fissure. Transversely it was about half an inch from the mesial surface (Dr. Hughlings Jackson, 'Trans. Clin. Soc.,' 1887).

shown by a case in which convulsions beginning in the toe were due to a cicatrix in this situation, and the removal of the part by Horsley left paralysis of the toe only.*

The arm centre (Fig. 11) appears to occupy the middle third of these convolutions, but extends higher up the ascending frontal than the ascending parietal, perhaps reaching almost to the edge of the hemisphere. A small lesion very near the longitudinal fissure has paralysed the arm. It is probable that this centre overlaps that for the leg.

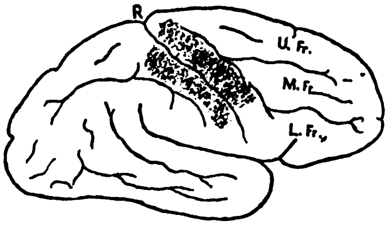

FIG. 11.—Position of the arm.

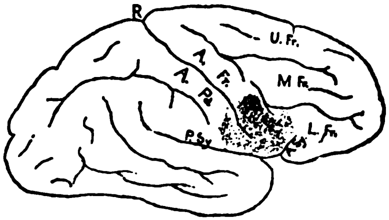

FIG. 12.—Position of the centre for the face and tongue.

The centre for the movement of the face (Fig. 12) lies in the lower third of the ascending frontal. It is probable, but not yet proved, that it extends on to the ascending parietal.† The centre for the movement of the angles of the mouth lies opposite the fissure between the middle and lower frontal convolutions.‡ The lips and tongue are apparently represented together in the lowest part of the ascending frontal, and perhaps in the adjacent root of the third frontal. The orbicularis oris and the transverse fibres of the tongue habitually act together; hence we cannot separate the centres for the face and tongue.§

We have no sure evidence at present as to the position of the centre for the movement of the head and eyes (said to occupy, in the monkey, parts of the first and second frontal); it must be of great importance. The effect of its activity determines our recognition of the relation of the body to external objects, especially to those that are seen. It is only the relation of these to one another that we can learn from the field of vision alone; the centre for the trunk muscles to be placed

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* Horsley, 'Int. Journal of Med. Science,' April, 1887.

† All parts of the face were paralysed by a lesion which involved the right of the second frontal and adjacent part of the ascending frontal, but below the cortex extended to the first and third frontal ('Mill. Journ. Nerv. and Mental Dis.,' 1887)

‡ See the conclusive case of Dr. Berkeley, of Baltimore, figured in the chapter on facial spasm.

§ It is probable that the movements of the jaw are also represented in this region. In the course of an operation on man, Horsley found that stimulation of the ascending frontal, at the junction of the middle and upper third of the facial region, caused a lateral movement of the jaw as well as of the angle of the mouth.

(according to the experiments of Horsley and Schäfer) on the inner surface of the hemisphere (see p. 14) still needs verification. The reason for this uncertainty has been already mentioned; compensation occurs so readily that the loss of movement quickly passes away.* It has been thought that there is a centre for the movement of the upper eyelid in the angular or lower parietal lobule (Landouzy and Grasset); an old lesion has been found in the angular gyrus in a case of ptosis of long duration.† Wernicke has concluded from Munk's researches that the neck region is represented in the part of the frontal lobe which lies in front of the middle of the anterior central convolution, and a case published by Fraenkel,‡ in which there was excessive rigidity of the neck muscles would seem to bear out this conclusion, as examination showed the existence of a large clot covering the foot of the second and the adjacent part of the third left frontal convolution immediately in front of the anterior central convolution.

There has been much discussion regarding the precise nature of these centres, and opinion is still divided on the matter. It is certain, however, that movements are produced by their stimulation in man as well as in animals.§ It is certain also that the fibres which conduct motor impulses to the cord spring from them, and pass directly downwards. Hence it is difficult to believe that any considerable amount of error can be involved in designating the region "motor," especially from the standpoint of practical medicine. We need not, therefore, conceive that these parts subserve no other function (we shall presently see reason for believing that they have sensory as well as motor functions). It is instructive, in this connection, to note that in this part are found the largest ganglion-cells met with in the cortex, cells comparable to, though exceeding in size, the certainly motor cells of the anterior cornu of the spinal cord.‖

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* It is in the highest degree probable that the position of the centres corresponds to that in the monkey, and this is supported by an interesting case recorded by Horsley ('Int. J. of Med. Science,' April, 1887), which is not, however, quite conclusive.

† Lemoine, 'Rev. de Méd.,' 1887. This harmonises with the sensory localisation, since the upper lid is raised for intent vision.

‡ 'Charité Annalen,' 1886.

§ First demonstrated by Bartholow, and since abundantly confirmed in the course of surgical procedure.

‖ A theory of the nature of the so-called motor centres, which has received wide acceptance in Germany, is that of Munk. This theory regards them as essentially sensory; the movements which result from their activity are due to the fact that the nerve processes in them represent "conceptions of movements" composed of the cutaneous and muscular sensations and sensations of active innervation. These are regarded as essentially sensory conceptions, and hence Munk terms this region the "Sense-sphere" (Fuhlsphäre). An objection to this view is that the "sense of innervation," one element of the threefold "conception of movement," is a sense of something which is not itself sensory, and which is commonly termed motor, and that destruction of this region causes loss of this "motor" function out of all proportion to any demonstrable sensory loss.

Sensory Centres.—Our knowledge of the position of the centres for sensation of the limbs and trunk is far less precise than is that of the motor centres. According to Flechsig the fibres of the sensory path of the internal capsule pass towards the outer surface of the cortex, towards the region which, roughly speaking, lies beneath the parietal bone, *i. e.* the central convolutions and the parietal lobe. The meagre facts of pathology, as far as they go, are in harmony with this view. One case is shown in Fig. 15. In another case complete left-sided loss of sensibility, including the eye and other special senses, resulted from a layer of softening at and just beneath the surface of the greater part of the convexity of the right hemisphere, the medial aspect and internal capsule being unaffected.* The view that the central convolutions have some sensory function, as well as a motor function, is supported by the facts that there is often slight blunting of sensibility on the extremity of a limb paralysed by disease in this region, and that in convulsions due to irritating lesions in this situation a sensory aura very often precedes the motor spasm. It is quite possible that the sensory region extends also to the medial surface of the hemisphere, just as does the motor region, but all the facts hitherto observed are opposed to the inference drawn by Ferrier from the experiments on animals, that any part of the medial surface has an exclusive or even a preponderant relation to cutaneous sensibility. One reason why we have so little evidence of the seat of this function is that extensive compensation is possible. Thus a unilateral lesion in childhood, however extensive, scarcely ever causes permanent loss of sensation.

Smell.—The indication of experiment is that there is a centre for smell at the anterior extremity of the uncinate convolution on the medial surface of the temporal lobe, related to the olfactory nerve of the same side.† This is supported, as regards man, by the facts that some fibres of the olfactory nerve can be traced towards this region, and that olfactory symptoms have been observed in a few cases of disease of this part. Thus epileptic fits beginning with an olfactory aura were associated in one case with softening in this region.‡ But it is probable that other fibres, or fibres from this centre, cross the middle line (perhaps in the anterior commissure) and go to some part of the cortex of the opposite hemisphere, since there are cases in which organic disease of one hemisphere has caused loss of the sense of smell, in addition to that of the other special senses, on the opposite side.§

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* Demange, 'Revue de Méd.,' May, 1883, p. 391. A collection of clinical facts pointing to the parietal and central regions as the chief sensory region is given by Allen Starr, 'Journal of Nervous and Mental Diseases,' 1884, p. 327.

† See case of Dr. Churton, Leeds, 'Brit. Med. Journ.,' 1887.

‡ McLane Hamilton, 'New York Med. Journal,' June, 1882; also Hughlings Jackson and Beevor, 'Brain,' vol. xii, p. 304.

§ Such a case, with autopsy, showing softening of the hinder part of the internal capsule, is recorded by Féré, 'Arch. de Neurologie,' 1885. I have seen several cases

Vision.—Numerous observations have established beyond question the fact that hemianopia results from disease of the occipital lobe, which thus constitutes a centre for the fibres from the same-named half of each retina, and receives impressions from the opposite half of each field of vision. The impressions from the retinæ reach it by the optic tract, and probably by the optic thalamus, and by fibres from the thalamus through the white substance of the occipital lobe. Fuller details of this path are given in the account of the origin of the optic nerves. We do not know the exact position of the visual centres in the occipital lobe. Hemianopia has resulted from disease of the apex of the lobe, the outer surface, and the medial surface, but in some cases of partial lesion the white fibres of the optic path may have been involved. Complete hemianopia has most frequently been produced by disease of the apex of the lobe, and especially of the cuneus. Munk believes that, in animals, there is a serial surface representation of the half-field in the occipital cortex, the anterior half corresponding to the upper quadrant and the posterior half to the lower quadrant. Since small cortical lesions cause incomplete hemianopia, it is probable that in man also there is such a projection of the field as Munk describes, but it is not probable that the projection occupies the whole occipital region.* There must be a representation of the colour fields in the cortex, distinct from that for light and objects, since colour hemianopia has been met with as an isolated symptom. (See "Diseases of

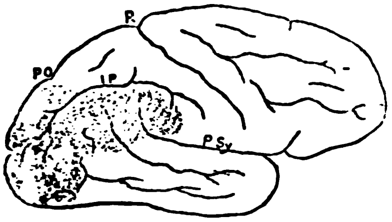

FIG. 13.—Cortical visual centres on the outer surface of the hemisphere. The darker shading indicates the region of the half-vision centre (the precise limitation of which is not yet known); the lighter shading is that of the supposed higher visual centre.

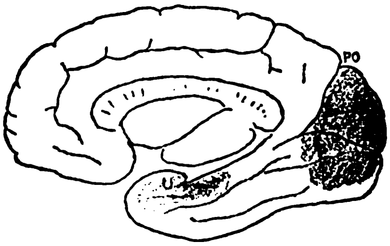

FIG. 14.—Inner aspect of the right hemisphere. Probable position of the visual centre in the occipital lobe and of the olfactory centre in the uncinate gyrus (U).

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during life in which smell was implicated. See also Fasola, 'Riv. Sper. di Fren.,' 1886; and Anderson, 'Brain,' vol. ix, p. 390.

* An important case has been recorded by Delépine ('Trans. Path. Soc.,' 1890). The patient had had right homonymous hemianopia for eight months, and contraction of the remaining fields. On the right side of the right field, however (which was otherwise blind), there was a persistent marginal circle of vision. *Post mortem* there was softening in the left cuneus and middle occipital convolution. The inner aspect of the cuneus was also affected, except the postero-superior portion, and the interesting question presents itself whether this part which escaped had a relation to the persistent marginal circle of vision in the right, otherwise blind, field.

the Optic Nerve.") Wilbrand* assumes that all impressions are conducted first to the apical region, because disease there causes total loss, and that there is a re-representation of the colour half-field in front of this.†

But disease sometimes causes, not hemianopia, but "crossed amblyopia," *i. e.* dimness of sight in the opposite eye, generally with concentric diminution of the field.‡ The theory which best explains the fact is that on the outer surface, in front of the occipital lobe, there is a higher visual centre in which the half-fields are combined, and the whole opposite field is represented. Such a centre, in animals, is localised by Ferrier in the angular gyrus (in which term he includes the extremity of the supra-marginal convolution). Pathological evidence in man points to some part of the same region as the seat of this centre. In the very few post-mortem examinations in cases with "crossed amblyopia" the posterior and inferior part of the parietal lobe, *i. e.* the angular gyrus, in its widest sense, has been involved in the disease. In the case mentioned on p. 20, recorded by Demange, the loss of sight of the left eye was an early symptom, and the softening of this region appeared older than that elsewhere. Another instructive case, recorded by Dr. Sharkey, is shown in Figs. 15 and 16. It would seem that the field of the same side is also represented in this centre, since the amblyopia in the opposite eye is usually accompanied by a slight restriction of the field of the eye on the same side. Thus the centre must be assumed to represent the whole of both fields, but that of the opposite side in a far higher degree than the field on the same side. This assumption enables us to understand another curious fact, viz. that the "crossed amblyopia" generally lessens after a time (while hemianopia is usually persistent). If the higher centre in each hemisphere is connected with both retinæ, it is conceivable that the loss caused by disease of one hemisphere may be compensated by the function of the other hemisphere. Hence we can understand that atrophy of this region, congenital or dating from early life, may be unaccompanied by any recognised loss of sight.§ The visual path will be considered in connection with the optic nerves.‖

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* 'Ophth. Beit. zur Diag. d. Gehirnkr.,' 1884.

† Varying impairment in extent of vision in a patient found to have glioma in angular gyrus (Anderson, 'Trans. Ophthal. Soc.,' 1890).

‡ I have seen several such cases, and others have been recorded, some by Ferrier ('Brain,' vol. iii, p. 456). Of course all ocular causes of amblyopia are excluded. (See chapter on Affections of Sight.)

§ As in a curious case recorded by Dr. Sharkey ('Med.-Chir. Trans.,' vol. lxvi, 1883, p. 293).

‖ Dr. Beevor has found that in the marmoset monkey there are no fibres from the corpus callosum to the cortex bounding the calcarine fissure. This part of the cortex is analogous to the visual centre in man, and if such an arrangement is also present there, it might throw some light on the persistence of hemiopia after lesion of this part.

Cortical lesions in dogs that do not cause loss of sight may abolish or impair the power of recognising the nature of seen objects, although they can be recognised at once when some other sense is employed; the condition has been termed mind-blindness (Munk*). The power thus lost seems to be subserved by structures in or near the higher visual centre, but the position of the lesion has not yet been ascertained in any case in which the loss was considerable and persistent. The loss of course includes the recognition of words, and the latter may occur without inability to recognise objects in general. Mind-blindness was produced by the lesion indicated in Fig. 17, but this case, while it agrees with the probable localisation, was too brief in duration to constitute actual proof of the seat of disease on which the symptom depends.† The subject is con-

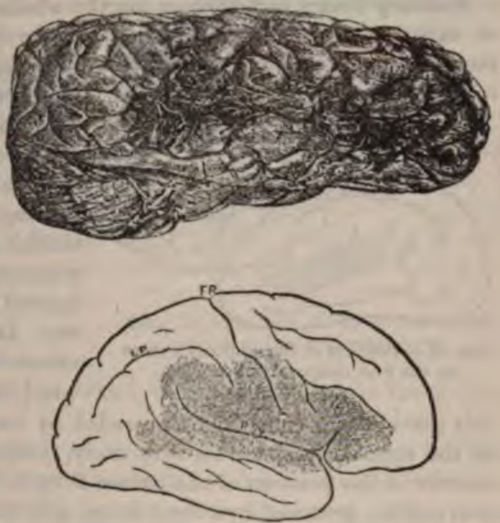

FIG. 15.—Aspect of right hemisphere, and FIG. 16 probable area of the lesion, in a case of embolism of the right middle cerebral artery (Sharkey, 'Med.-Chir. Trans.,' 1884, p. 265). The immediate effect of the lesion was left hemiplegia and hemianæsthesia, with almost complete blindness of the left eye, and loss of hearing and taste on the left side. Four weeks after the illness there was marked improvement in the special senses, and, a little later, in the hemianæsthesia. Six and a half weeks after the onset the special senses were normal, the leg had regained much power, and a fortnight later sensation was normal. The paralysis of the arm continued until death, seven years later.

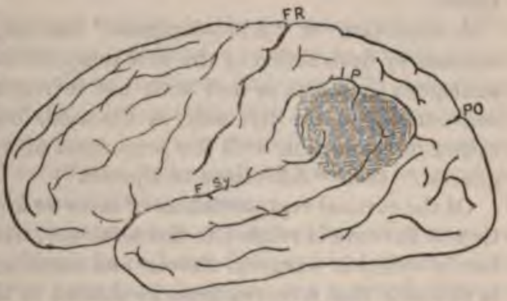

FIG. 17.—Position of lesion in the left hemisphere (angular gyrus), which caused complete mind-blindness during the short time the patient lived. (Chauffard, 'Rev. de Méd.,' 1881, p. 940.)

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* 'Deut. med. Wochensch.,' 1877, No. 13; and 'Arch. f. Anat. u. Phys.,' 1878, p. 162.

† The same statement applies to a case of depressed fracture of the skull in this region, in which mind-blindness existed for two weeks (McEwen, 'Brit. Med. Journal,' Aug., 1888).

sidered further in the sections on "Affections of Speech," and "Diseases of the Optic Nerves." The theory remains the most probable.

Auditory Centre.—Pathology on the whole supports the indication of experiment, which places the auditory centre in the middle of the first temporo-sphenoidal convolution (Fig. 18). This convolution has been found atrophied in cases of congenital deafness.* Destruction of this region has been accompanied by loss of hearing in the opposite ear, although the loss has not been permanent. An instance is the case figured on the last page (Fig. 15). In a case under my observation of extensive tumour, in which the oldest part was beneath this convolution, convulsions preceded by an auditory aura, referred to the opposite ear, were a very early symptom. In another case a tumour in the position of the letters P Sy in Fig. 18 caused unilateral convulsions, preceded by a loud noise, as of machinery. The convolution on each side would appear, therefore, to be related to the opposite auditory nerve. It is important to note that the loss is not permanent, and this may explain another fact, that, in many cases in which the symptoms lasted for some time, the absence of deafness has been noted during life, although this convolution was found destroyed after death.† Hence it would seem that perfect compensation is possible, presumably by the corresponding centre of the opposite side. The complete deafness present at first in the case shown at Fig. 15 passed away completely at the end of six weeks. Thus each auditory nerve must be structurally connected with both hemispheres, although only the connection with the opposite hemisphere is habitually in functional action.

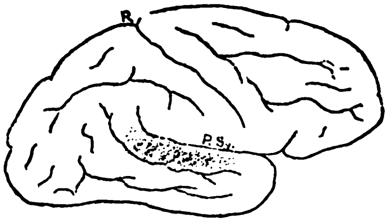

FIG. 18.—Position of the auditory centre in the first temporal convolution.

A condition of "mind-deafness" has apparently been caused in animals by destruction of the first temporal convolution (Munk). An analogous condition is met with not infrequently in man when this convolution on the left side is diseased, but it has been observed rather in connection with the perception of words than of sounds in general. (See "Affections of Speech.")

Of the cortical representation of taste we know nothing. The perception of flavours is related to the olfactory, not to the gustatory nerve; but in epileptic warning, flavour and smell are always distinct, and it is probable that a rearrangement obtains in the cortex.

Speech.—The relation of certain parts of the cortex to speech processes can be better understood when these processes have been considered in detail. The centres concerned in articulate speech are in the posterior extremity of the lowest frontal convolution, and the adjacent part of the ascending frontal, in the left hemisphere. They correspond, in part at least, to the centres for the movements (lips and tongue) by which articulation is effected. The perception of heard words is subserved by the centre for hearing in the first temporal, or by structures adjacent to it. There may be loss of the power of understanding heard words without deafness. The comprehension of seen words is a difficult and complex subject, which will be considered in the chapter on affections of speech.

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* Fletcher, Beach, and others.

† As in a case of softening recorded by Ball, 'New York Arch. of Med.,' April 1881.

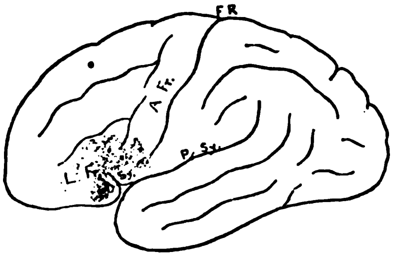

FIG. 19.—Position of the motor speech region in the left hemisphere.

Psychical Processes.—It is presumed that mental processes are subserved by those parts of the cortex that have no known motor or sensory function, and especially by the pre-frontal lobes. Many cases are on record in which considerable mental change was produced by extensive disease in this part, especially great when the disease was bilateral. Small lesions, however, may cause no symptoms, perhaps because there is considerable capacity for functional compensation. It would probably, however, be wrong to regard mental processes as exclusively related to the parts which are not known to have other functions, since the motor and sensory regions must also subserve mental operations.

THE CONNECTING TRACTS, CENTRAL GANGLIA, &C.

The next subject of medical importance is the course of the fibres that unite the various parts, and establish a connection with the spinal cord. The course of these fibres has been only partially unravelled, and the difficulty of the task is increased by the fact that many of the tracts are interrupted in places by grey matter. This interruption arrests the progress of secondary degeneration, which is so great an aid in tracing the course of interlacing fibres.

The centrum ovale of the hemisphere consists of medullated nerve-fibres, of which three classes have been distinguished according to their course:—(1) Fibres that pass between and connect different convolutions in the same hemisphere; amongst these is a special set which passes between the occipital and frontal lobes, in the inner part of the hemisphere, crossing the fibres to the corpus callosum, in the congenital absence of which it becomes very distinct.* It may subsidered further in the sections on "Affections of Speech," and "Diseases of the Optic Nerves." The theory remains the most probable.

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* Burdach, Meynert, Onufrowicz ('A. f. Psych.,' xviii).

Auditory Centre.—Pathology on the whole supports the indication of experiment, which places the auditory centre in the middle of the first temporo-sphenoidal convolution (Fig. 18). This convolution has been found atrophied in cases of congenital deafness.* Destruction of this region has been accompanied by loss of hearing in the opposite ear, although the loss has not been permanent. An instance is the case figured on the last page (Fig. 15). In a case under my observation of extensive tumour, in which the oldest part was beneath this convolution, convulsions preceded by an auditory aura, referred to the opposite ear, were a very early symptom. In another case a tumour in the position of the letters P Sy in Fig. 18 caused unilateral convulsions, preceded by a loud noise, as of machinery. The convolution on each side would appear, therefore, to be related to the opposite auditory nerve. It is important to note that the loss is not permanent, and this may explain another fact, that, in many cases in which the symptoms lasted for some time, the absence of deafness has been noted during life, although this convolution was found destroyed after death.† Hence it would seem that perfect compensation is possible, presumably by the corresponding centre of the opposite side. The complete deafness present at first in the case shown at Fig. 15 passed away completely at the end of six weeks. Thus each auditory nerve must be structurally connected with both hemispheres, although only the connection with the opposite hemisphere is habitually in functional action.

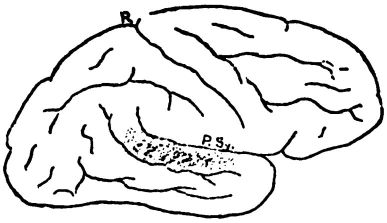

FIG. 18.—Position of the auditory centre in the first temporal convolution.

A condition of "mind-deafness" has apparently been caused in animals by destruction of the first temporal convolution (Munk). An analogous condition is met with not infrequently in man when this convolution on the left side is diseased, but it has been observed rather in connection with the perception of words than of sounds in general. (See "Affections of Speech.")

Of the cortical representation of taste we know nothing. The perception of flavours is related to the olfactory, not to the gustatory nerve; but in epileptic warning, flavour and smell are always distinct, and it is probable that a rearrangement obtains in the cortex.

Speech.—The relation of certain parts of the cortex to speech processes can be better understood when these processes have been considered in detail. The centres concerned in articulate speech are in the posterior extremity of the lowest frontal convolution, and the adjacent part of the ascending frontal, in the left hemisphere. They correspond, in part at least, to the centres for the movements (lips and tongue) by which articulation is effected. The perception of heard words is subserved by the centre for hearing in the first temporal, or by structures adjacent to it. There may be loss of the power of understanding heard words without deafness. The comprehension of seen words is a difficult and complex subject, which will be considered in the chapter on affections of speech.

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* Fletcher, Beach, and others.

† As in a case of softening recorded by Ball, 'New York Arch. of Med.,' April 1881.

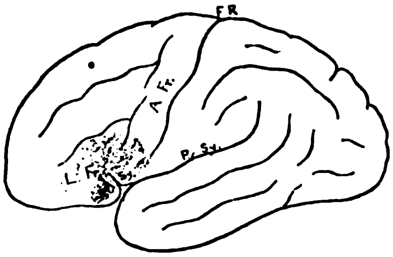

Fig. 19.—Position of the motor speech region in the left hemisphere.

Psychical Processes.—It is presumed that mental processes are subserved by those parts of the cortex that have no known motor or sensory function, and especially by the pre-frontal lobes. Many cases are on record in which considerable mental change was produced by extensive disease in this part, especially great when the disease was bilateral. Small lesions, however, may cause no symptoms, perhaps because there is considerable capacity for functional compensation. It would probably, however, be wrong to regard mental processes as exclusively related to the parts which are not known to have other functions, since the motor and sensory regions must also subserve mental operations.

The Connecting Tracts, Central Ganglia, &c.

The next subject of medical importance is the course of the fibres that unite the various parts, and establish a connection with the spinal cord. The course of these fibres has been only partially unravelled, and the difficulty of the task is increased by the fact that many of the tracts are interrupted in places by grey matter. This interruption arrests the progress of secondary degeneration, which is so great an aid in tracing the course of interlacing fibres.

The centrum ovale of the hemisphere consists of medullated nerve-fibres, of which three classes have been distinguished according to their course:—(1) Fibres that pass between and connect different convolutions in the same hemisphere; amongst these is a special set which passes between the occipital and frontal lobes, in the inner part of the hemisphere, crossing the fibres to the corpus callosum, in the congenital absence of which it becomes very distinct.* It may subserve movement of the head and eyes to the opposite side when a visual impression passes thence to the brain. (2) Fibres that pass inwards to the corpus callosum, and for the most part connect corresponding regions in the cortex of the two hemispheres.* Probably the fibres between the leg centres are at the back of the corpus callosum, those between the arm and trunk centres in the middle, and those between the head and eye centres in the fore-part.† (3) Fibres that pass to the central ganglia or crus cerebri. The last-named converge from all parts of the cortex to the inner and lower region of the hemisphere, where the central ganglia lie and the crus leaves the brain. If traced upwards from the crus and ganglia they radiate towards the cortex, and the radiation of the two hemispheres has been compared to a crown, and termed the "corona radiata." This expression is not now much used, but a limited application of the term is current and convenient: special groups of these fibres are termed "radiations," as the "pyramidal radiation," "optic radiation," &c.; while the fibres that thus radiate are sometimes termed "coronal."

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* Burdach, Meynert, Onufrowicz ('A. f. Psych.,' xviii).

The crus cerebri, it will be remembered, enters the inner side of the hemisphere, spreading out beneath the optic thalamus and caudate nucleus, and its fibres ascend in a layer beneath these ganglia on the inner side and the lenticular nucleus on the outer side. This layer is termed the "internal capsule," because it bounds internally the lenticular nucleus. In a horizontal section through these ganglia (Fig. 20) it is seen that the anterior and posterior parts of the capsule have not quite the same direction; the anterior part, between the body of the caudate nucleus and the fore-part of the lenticular nucleus, joins at an angle the posterior part between the optic thalamus and the hinder part of the lenticular nucleus. These are called the anterior and posterior

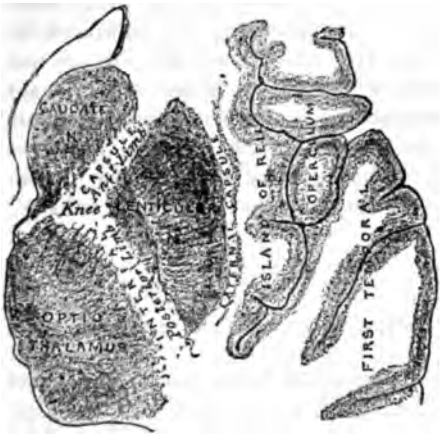

FIG. 20.—Diagram of horizontal section through the central ganglia and internal capsule.

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* This opinion has been confirmed by almost all modern investigations, with the exception of those of Dr. D. J. Hamilton ('Journ. of Anat.,' xix, p. 385), whose theory is scarcely to be reconciled with the certain facts of pathology, and whose evidence, from comparative anatomy, has been contested by Beevor ('Brain,' 1886), while the older view has been experimentally confirmed by Schäfer and Mott ('Brain,' viii, 1890). Stimulation of the surface of the corpus callosum excited the motor centres of both hemispheres; of one only when they had been destroyed in the other.

† Schäfer and Mott, loc. cit.

"limbs" of the capsule, and the angle at which they join is called its "elbow" or "knee."* In an experimental investigation into the excitability of the fibres of the internal capsule in the monkey,† Beevor and Horsley found that the fibres were arranged from before backwards in the same order as the foci of representation in the excitable part of the cortex taken along lines drawn at right angles to the direction of the fissure of Rolando; that the arrangement of fibres in the different segments of each great division was the same as prevails in the cortex, and that the character of each movement was represented in the capsule as in the cortex. The extent of the lenticular nucleus from front to back corresponds to both the caudate nucleus and optic thalamus together, but the slender tail of the caudate nucleus extends as far back as the posterior extremity of the lenticular nucleus, curving down into the roof of the descending cornu of the lateral ventricle. The two parts of the corpus striatum (caudate and lenticular nuclei) are connected at their anterior and posterior extremities by slender tracts of grey matter, which pass between the fibres of the capsule; elsewhere these fibres separate the two ganglia. In the lenticular nucleus, as seen in transverse section, three parts can be distinguished by a difference in tint—the inner, middle, and outer, or first, second, and third, the last being by far the largest (see p. 41).

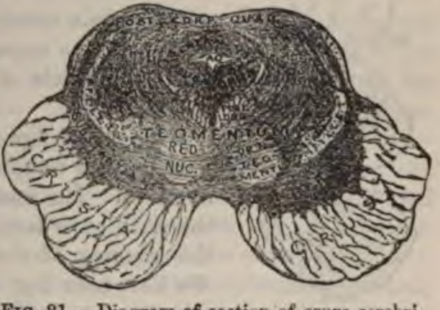

FIG. 21.—Diagram of section of crura cerebri. AQ, aqueduct of Sylvius.

A section through the crura cerebri above the pons (Fig. 21) shows them connected above by the corpora quadrigemina. The crus proper is separated into two parts, anterior (ventral) and posterior (dorsal),‡ by the "locus niger;" the lower or anterior is called the *crusta*, the upper or posterior the *tegmentum*. The crusta is also called the "pes" or "basis," and the former ("Fuss") is the term usually employed in Germany; but neither of these words lends itself to the English system of terminology, and it is better to use the older term "crusta" and its adjective "crustal." The tegmentum is much greyer than the crusta, because it contains many nerve-cells mingled with the fibres, while the crusta consists almost exclusively of nerve-fibres. The tegmentum varies in tint in different parts of its section. Near the middle line is a round or oval area, greyer than the rest, and often reddish grey. It is the "red nucleus," or "tegmental nucleus." To the outer side of the red nucleus, and just above the outer part of the locus niger, a paler curved band is seen, largest at its inner extremity. This is the *fillet*, an important tract of fibres, which extends up from the lowest part of the pons, and occupies there also the same relative position, the lowest part of the tegmentum. Above the red nucleus is a small triangular white area, near the middle line, the section of a compact bundle of "posterior longitudinal" or "posterior horizontal" fibres.

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* Such a bend (as in a pipe) is termed a "knee" in Germany, an "elbow" in this country. It is perhaps better to term the junction the "angle" of the capsule.

† 'Phil. Trans.,' 1890.

‡ Although the direction of the pons and crura is nearer the horizontal than the vertical, it is convenient to retain the terms anterior and posterior in the same significance as in reference to the cord—the more so because the fibres from the crus again assume an ascending course when they enter the hemisphere.

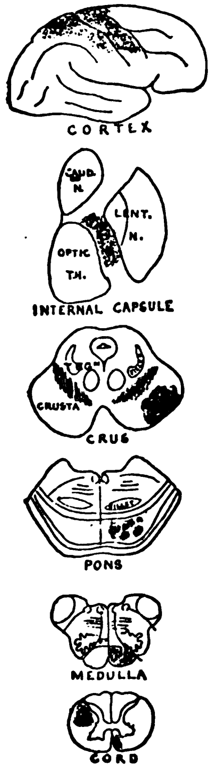

FIG. 22.—Diagram of the course of the pyramidal tract of the right hemisphere.

In a section through the middle of the pons (Fig. 27, p. 33) the distinction between the two parts, crustal and tegmental, is still indicated by the position of the fillet in the lowest layer of the tegmentum. The crustal portion below (*i. e.* in front of) the fillet is here increased in bulk by the transverse fibres from the middle peduncle of the cerebellum and by much scattered grey matter. In the tegmentum the posterior longitudinal bundles are still seen near the middle line and close to the floor of the fourth ventricle. Between them and the fillet is an area consisting of interlacing bundles of fibres, transverse and longitudinal, the "reticular formation." In the tegmental part of the pons there are also various collections of grey matter from which certain cranial nerves take origin. These lie chiefly in the upper part, near the floor of the fourth ventricle, and will be subsequently described.

Our knowledge of the course of the motor path is more complete than that of any other set of fibres. The relations of the spinal portion of this path have been described in the account of the anatomy of the spinal cord (vol. i, p. 172). We have seen that it there occupies the two pyramidal tracts, anterior or direct, and lateral or crossed. The precision with which their limits are indicated by secondary degeneration enables us to trace their course with equal certainty in the brain. The degeneration has revealed also the remarkable fact that the pyramidal fibres extend fron the central convolutions to the spinal cord without any interruption by grey matter. Tracing their course up from the cord (Fig. 22), we find that each lateral tract crosses the middle line in the medulla, and, uniting with the anterior or direct tract, forms the "anterior pyramid;" from this the name is taken by which the fibres are known throughout their course. Entering the pons the two pyramids are covered by the superficial layer of transverse fibres, and divide into a series of bundles. Thus divided, they course up through the crustal portion of the pons, lying between the superficial and deep layers of transverse fibres, and surrounded by much grey matter, with which, however, their fibres have no connection. Above the pons the bundles again unite, and in the crus cerebri the pyramidal fibres lie together, and occupy the middle two fifths of the crusta, extending from the surface below almost, but not quite, to the substantia nigra above. Passing beneath the optic thalamus, they ascend between it and the lenticular nucleus, as part of the internal capsule. They occupy the anterior two thirds of its posterior limb, the hinder third containing the sensory path.* Above the lenticular nucleus they radiate through the white substance of the hemisphere to that part of the cortex in which experimental stimulation causes movement in the limbs, viz. the two central convolutions with the superior parietal lobule and paracentral lobule. The motor impulses originating in these convolutions appear to be conducted directly to the grey matter of the spinal cord by these fibres, without the intervention of any nerve-cells, either of the central ganglia or the pons. If these convolutions are destroyed, the fibres degenerate down to the lowest part of the cord. Some fibres from the motor cortex pass into the narrow "external capsule," outside the lenticular nucleus, into which their degeneration can be traced. Their further course is unknown.

The "motor" convolutions contain also centres for the movements exerted through some of the motor cranial nerves—movements of the jaw, face, and tongue. The fibres from these centres pass from the cortex with the pyramidal fibres. The centres for the face and tongue are lowest in the cortex (as we have seen), and their path lies correspondingly below that of the limbs in the centrum ovale. But the change from the vertical to an antero-posterior relation, in the internal capsule, brings the fibres for the face and tongue in front of those for the limbs, and they occupy the bend or angle of the capsule (Fig. 23). In the crusta they occupy a corresponding position on the inner (medial) side of the pyramidal fibres (Brissaud, Raymond and Artaud), the change in direction of the crus having brought to the inner side that which was anterior in the capsule and inferior in the cortex. It is probable that, in the pons, the fibres for the cranial nerves continue with the pyramidal fibres, lying on the inner side of the latter; hence the face and limbs may be paralysed together by a small lesion in the upper part of the pons. When these fibres approach the level of their nuclei, they leave the neighbourhood of the pyramidal tract, and, crossing the middle line, pass to the nucleus from which the cranial nerve takes origin.

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* Perhaps not exclusively, however. A few fibres of the motor path have been found in the hinder third (Mannkopf, 'Zeitsch. f. kl. Med.,' 1884, Sup.). It is possible, however, that these fibres belong, not to the pyramidal tract, but to the fillet (q. v.).

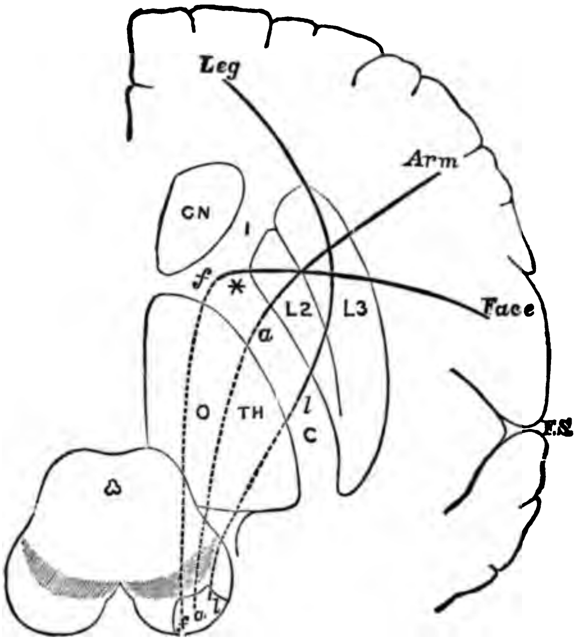

Fig. 23.—Diagram to show the relative position of the several motor tracts in their course from the cortex to the crus. The section through the convolutions is vertical; that through the internal capsule, I C, horizontal; that through the crus is again vertical; C N, caudate nucleus, O TH, optic thalamus, L 2 and L 3, the middle and outer parts of the lenticular nucleus; *f*, *a*, *l*, face, arm, and leg fibres. The words in italics indicate the corresponding cortical centres.

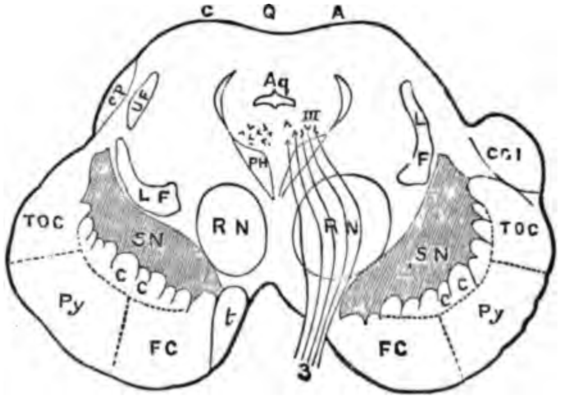

Fig. 24.—Diagram of section of the crus (modified from Wernicke). L F, U F, upper and lower fillet; C Q A, anterior corp. quad.; Aq, aqueduct; III, nucleus of third nerve (3); P H, posterior horizontal fibres; c p, brachium of the post. corp. quad.; R N, red nucleus; S N, substantia nigra; C G I, internal geniculate body; T O C, temporo-occipital cerebellar fibres; Py, pyramidal fibres; F C, fronto-cerebellar fibres; C C, caudate cerebellar fibres; t, inner fibres of crusta to tegmentum.

Thus, of the white fibres that constitute the crusta, or lower half of the crus cerebri, less than half belong to the pyramidal tracts. What are the other crustal fibres? They lie partly on the outer side of the pyramidal fibres (T O C, Fig. 24), partly on the inner (medial) side (F C), partly above (C C), between the pyramidal fibres and the substantia nigra. Only those on the inner (medial) side of the pyramidal tract pass up into the internal capsule. The change in the direction of the crus brings these inner fibres in front of those of the pyramidal tract, and they form the anterior limb of the capsule (Fig. 25). They radiate to the cortex of the "pre-frontal lobe," *i. e.* that part of the brain which lies in front of the central convolutions. From the crus they pass downwards into the pons. They degenerate downwards, and

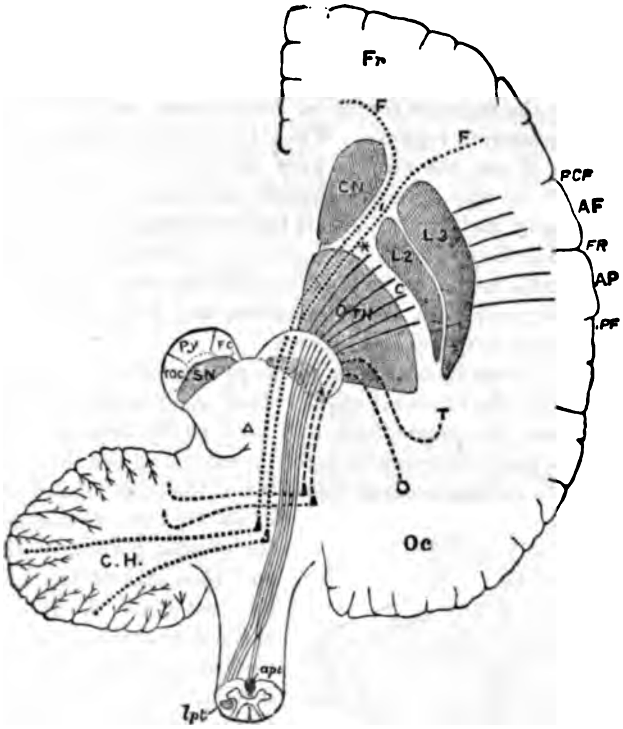

FIG. 25.—Diagram of the course of the motor tract as shown in a diagrammatic horizontal section through the cerebral hemisphere, pons, and medulla. Fr, frontal lobe; Oc, occipital lobe; A F, ascending frontal, and A P, ascending parietal, convolutions; *P C F*, pre-central fissure in front of the ascending frontal convolution; *I P F*, interparietal fissure. A section of the crura is lettered on the left side: S N, substantia nigra; Py, region occupied by the pyramidal fibres (motor tract), which on the right are shown as continuous lines, converging in the white substance of the hemisphere, to pass through the posterior limb of I C, the internal capsule (the elbow of which is shown at *)—through the crus and pons, and to divide in the medulla into the decussating lateral pyramidal tract (*lpt*) and the direct anterior pyramidal tract (*apt*).

have been found degenerated in many cases of disease of the anterior part of the capsule and in extensive softening of the cortex.* Hence we may assume that they conduct downwards, but their degeneration stops in the pons, apparently because they end in the grey matter which is so abundant in the crustal portion. This grey matter also receives fibres from the middle cerebellar peduncles, and these fibres probably decussate in the middle line before entering the nerve-cells (Lallemont). They seem to continue the path constituted by the inner crustal fibres. Thus each pre-frontal lobe is specially connected, not only, as we have seen, with the occipital cortex of its own side, but also with the opposite cerebellar hemisphere, and chiefly with its lateral and posterior regions. When the cerebellum is congenitally absent, these fibres, the crustal grey matter of the pons, and the anterior limb of the internal capsule are absent (Flechsig). In animals the grey matter atrophies if the cerebellum is excised.†

One small bundle of fibres in the inner part of the crusta (t, Fig. 24), lying close to the medial surface, differs from the rest. As it descends, it passes backwards into the tegmentum, and joins the fillet. Its further relations have not been traced.

The crustal fibres that lie outside the pyramidal tract‡ (T-O-C, Fig. 25) do not enter the internal capsule. They leave the other fibres when the crus enters the hemisphere, and pass partly beneath the lenticular nucleus, partly between its posterior extremity and the outer geniculate body, to radiate towards the cortex of the occipital and temporal lobes. Downwards they pass into the crustal portion of the pons and then end in the grey matter, just as do the inner fibres, and are probably, in like manner, connected with the cerebellar hemisphere, chiefly, Flechsig thinks, with the upper surface, near the middle lobe. These fibres are also wanting when the cerebellum is absent. Flechsig thought that they do not degenerate downwards, but it appears certain that they do, since they have been found degenerated in several cases of disease of the occipital and temporal cortex.§ It was formerly believed

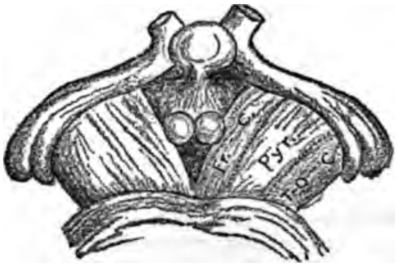

FIG. 26.—Crura cerebri; position of the fronto-cerebellar (Fr-C), pyramidal (Pyr), and temporo-occipital cerebellar fibres (T-O-C) on the surface.

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* Rossolymo, 'Neur. Cent.,' 1886.

† Marchi, 'Rivist. sperim. de freniat.,' 1886.

‡ Sometimes called "Türck's bundle," but to be carefully distinguished from Türck's column" in the cord.

§ Bechterew, 'Cent. f. Nervenh.,' 1886, p. 635. See also under "Secondary Degenerations;" also Kreuser ('Allg. Zeitschr. f. Psych.,' xlviii) relates a case in which there was absence of the occipital lobe on one side, and amongst other changes that these fibres enter the internal capsule and constitute its posterior extremity, which is known to be the chief sensory path, and this statement is still made in some text-books. But Flechsig, by developmental investigations, has shown that this is an error, and his conclusions have been confirmed by pathology. As the fibres separate from the pyramidal tract, their place is taken by sensory fibres from the tegmentum, and thus the extremity of the capsule is formed.

The last part of the crusta to be considered is the thin layer of fibres which lies above the pyramidal tract, between it and the substantia nigra. According to Flechsig, these arise, above, from the corpus striatum (caudate nucleus, and outer part of the lenticular nucleus), and they seem to end, below, in the pons, probably in the crustal grey matter, as do the other fibres just described. They degenerate downwards, and cannot be traced beyond the pons. They may thus connect the corpus striatum and cerebellum, in the same way as the inner group connects the pre-frontal lobe and cerebellum. Some of them may be connected with the grey matter of the substantia nigra.

Thus, of all the fibres of the spinal cord, the pyramidal tracts alone find a place in the crusta of the cerebral peduncle. It may be noted, moreover, that these are the only long spinal fibres that degenerate downwards. The other fibres of the cord are either short fibres or degenerate upwards, and are connected with the cerebellum or with the tegmentum of the crus. Their connections are at present very imperfectly known, but are of great importance. The sensory path must be sought in some of these fibres; it certainly lies in the tegmentum of the crus, and probably passes to the crus by the tegmental region of the pons.

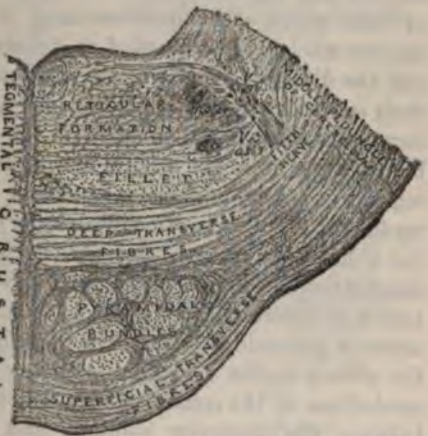

FIG. 27.—Diagram of one half of a section through the middle of the pons. *S o*, superior olivary body; *V as*, ascending root, *V m*, middle nucleus, *V motor*, motor nucleus of the fifth nerve.

In the tegmental region of the pons, as we have already seen, three chief groups of fibres may be distinguished, (Fig. 27), the small bundle of "posterior longitudinal fibres," the "fillet," and the extensive "reticular formation." We have to consider the connection of the remaining tracts of the cord with these structures and with the cerebellum, and also the relation of these tegmental fibres to the cerebral hemispheres.

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there was descending degeneration in the lateral part of the crus, which could be followed down to the pons, where it gradually disappeared.

which spring from the nucleus. Fibres also pass from the lateral nucleus and reticular formation to the restiform body and cerebellum, but the direction in which they conduct is uncertain. Flechsig thinks that they may conduct from the cerebellum to the reticular formation. Besides these two sets of fibres, the restiform body contains also the fibres from the opposite olivary body to the cerebellum, which we have already considered.

The antero-lateral ascending tract seems to pass to the reticular formation, but its fibres are most likely interrupted by the grey matter of the lateral nucleus. We have seen that the posterior columns are also connected with the reticular formation. It is probable that the upward sensory path passes, in part at least (and perhaps chiefly), by this structure, which can be traced through the pons into the tegmentum of the crus, in which the sensory path certainly lies.

The *fillet* or *lemniscus*, as we have seen, is a layer of fibres that lies between the crustal and tegmental portions of the pons, on the anterior (ventral) side of the reticular formation. It extends through the crus cerebri, where, however, it moves outwards, and lies in the outer part of the tegmentum. There is still much difference of opinion regarding the connections of its fibres, in spite of the fact that these have been the subject of an immense amount of recent investigation.* It is clear, however, it contains several different sets of fibres. Below, the fillet arises, as already stated (p. 34), in the interolivary layer, formed of the arciform fibres which proceed from the nuclei of the opposite posterior columns, median and external; part of the fillet thus appears to be a continuation of the path of these columns, and is conjectured by Spitzka to conduct chiefly centripetal impressions from the muscles. Many of the fibres, however, degenerate downwards; others seem to degenerate upwards. In a case in which the fillet was destroyed on the left side of the pons by an old hæmorrhage, Spitzka traced the descending degeneration through the interolivary layer to the opposite nuclei of the posterior columns. But in another case of focal lesion an extensive ascending degeneration of the same part of the fillet was found by Meyer.† A few fibres also seem to pass to the lateral column of the cord. Among the different sets of fibres in the fillet we may distinguish the following upward connections:—(1) Many fibres pass by the red nucleus, and are associated with fibres from this and from the opposite cerebellar peduncle, in what is termed the "lenticular loop," a set of fibres which, beneath the optic thalamus, turn outwards and pass transversely through the internal capsule, to end in the lenticular nucleus* (Fig. 29). (2) Some fibres pass, in the pons, into the reticular formation. (3) Some fibres go to the posterior corpus quadrigeminum. Some of these end below in the superior olivary body. (4) Other fibres end in a collection of grey matter lying outside the junction of the two corpora quadrigemina, the "nucleus lemnisci" of Flechsig and Bechterew. (5) Fibres pass up to the posterior part of the internal capsule, and radiate with these capsular fibres to the central and parietal cortex. Most of them are said to be connected with the posterior median nucleus. It appears probable, however, that they degenerate downwards (Monakow, Spitzka).

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* The most important recent writings on the subject are those of Flechsig ('Plan des Menschlichen Gehirns'); Flechsig and Bechterew (abstract by Flechsig in the 'Neurologisches Centralblatt, 1885, p. 356); Monakow (ib., p. 265); Wernicke ('Gehirnkrankheiten,' Bd. i); Spitzka ('New York Med. Record,' 1884, Nos. 15—18); and A. Bruce ('The Mid and Hind Brain,' 1892).

† 'Arch. f. Psych.,' xvii, p. 489.

The *reticular formation* contains numerous longitudinal fibres, the downward connection of which, with the lateral column and posterior columns, has been already described. It also receives fibres from the nerve-nuclei of the pons. At the upper part of the pons it rapidly lessens in size, and many of its fibres go to the upper corpus quadrigeminum,† and from this again fibres proceed to the posterior part of the internal capsule and tegmental radiation. The fibres that do not go to the corpus quadrigeminum pass upwards to the optic thalamus, and perhaps to the posterior part of the internal capsule and tegmental radiation; a few go to the grey matter lining the third ventricle.

The third group of longitudinal fibres in the tegmental portion of the pons is that termed the *posterior longitudinal fibres*, lying near the posterior surface and middle line (Figs. 24, 29, 39). This group contains fibres of different size. The finer are continuous below with some of those of the anterior column of the cord, and above pass to the central grey substance that lines the third ventricle (Flechsig). The coarser fibres extend only from the nucleus of the third and fourth nerves above to the level of the nucleus of the sixth below, perhaps also of the seventh,‡ and connect these nuclei. Some of the fibres decussate, and they no doubt subserve the complex associated action of the eyeball muscles, their relation to which will be considered later.

In the lower part of the pons, above the olivary body, is a small body, with somewhat sinuous outline, the *superior olivary body*. It lies in the anterior part of the tegmentum, and its important connections have been traced by Bechterew.§ According to him, fibres pass up from it to the fillet and central grey substance of the posterior quadrigeminal bodies; other fibres pass to the nucleus of the sixth nerve, the anterior auditory nucleus, the cerebellum (roof nucleus in the middle lobe), and to the lateral column of the spinal cord. This connection suggests that the body has important central functions, and it may be through it that an auditory impression causes a lateral movement of the eyes and head, the former through the nucleus of the sixth, the latter through the lateral column of the cord. The connection with the cerebellum may be one path by which disease or stimulation of the cerebellum influences ocular movements. The corpora quadrigemina are probably concerned in the movements of the eyes, and the fibres from them to the superior olivary body belong to that part of the fillet which, according to Flechsig, degenerates and conducts downwards.

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* In a case of absence of the cerebellum, examined by Flechsig, the red nucleus and fibres from the superior cerebellar peduncle were absent, and the fillet fibres in the lenticular loop were very distinct.

† A few of these reticular fibres, that lie close to the fillet, are by some authorities regarded as belonging to this structure.

‡ See Turner and Tooth, 'Brain,' 1892, and Mendel, 'Neur. Cent.,' 1887, p. 537.

§ 'Wratsch,' No. 32, 1885; 'Cent. f. Nervenheilk.,' 1886, p. 587.

Beneath the corpora quadrigemina the tegmentum receives a considerable accession of fibres from the passage into it of the superior cerebellar peduncle, the fibres of which come chiefly from the dentate nucleus. They may be connected, through this, with the fibres from the olivary body, and also with the cortex of the cerebellum. In the tegmentum these fibres cross the middle line, and are connected with the opposite red nucleus, which is absent, with the fibres of this peduncle, in congenital absence of the cerebellum (Flechsig). Thus the red nucleus may be connected with the olivary body of the same side by means of the opposite dentate nucleus. Proceeding upwards from the red nucleus, the fibres divide into two groups: one passes forwards and outwards to the lenticular loop, and thus to the lenticular nucleus (Fig. 29); the other courses backwards and outwards, partly into the basal part of the optic thalamus, partly into the posterior part of the internal capsule and tegmental radiation. Those fibres

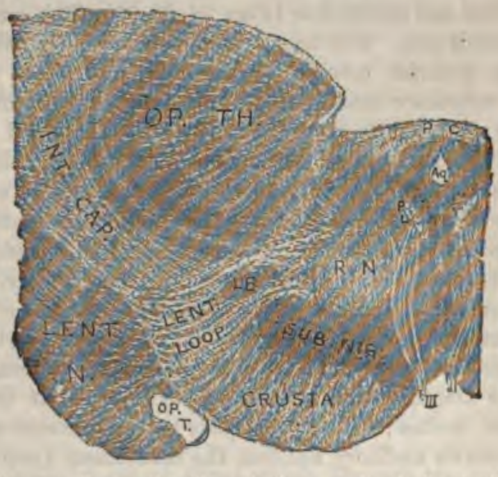

FIG. 29.—Diagram of a section through the crus, &c., in front of the corp. quad. P C, posterior commissure; Aq, aqueduct of Sylvius; P L, posterior longitudinal fibres; III, third nerve; L B, Luy's body; OP T, optic tract. (Modified from Wernicke.)

which enter the thalamus perhaps merely pass through it to the capsule.

Thus the chief destination of the tegmentum, including the superior cerebellar peduncle, is twofold—the lenticular nucleus, and the cortex of the hemisphere by the tegmental radiation. Fibres go to each of these from the fillet and the cerebellar peduncle, and other fibres of the fillet, together with those of the reticular formation, ultimately reach the tegmental radiation. As before stated, the sensory path certainly lies in the tegmental radiation where this begins in the hindmost region of the internal capsule. It is also practically certain that most forms of sensation from the limbs pass through the tegmentum of the pons, and it is probable that the path is different for the different forms of sensibility. It may be assumed that no sensory impressions pass in the larger division of the fillet (which is connected above with the lenticular nucleus) since the fibres degenerate downwards, nor by the posterior longitudinal bundles, which are not connected with the tegmental radiation. There remain, then, as possible paths only the upper smaller division of the fillet and the longitudinal fibres of the reticular formation. It has been conjectured that some forms of sensibility, *e. g.* visceral sensibility and muscular sensibility, may pass through the cerebellum and superior cerebellar peduncles. But the perceptions of posture and movement are very complex. They depend in part on afferent impression from the muscles, tendons, ligaments, and joints, which are perceived as sensations, or can be so perceived by attention. The path of these is probably through the pons, and some facts recently ascertained suggest that they pass up in that part of the reticular formation and fillet which is nearest the raphe.* But the perceptions of posture and movement are probably also due in considerable degree to the activity of the motor cells of the cortex, under the influence of impulses that proceed from the middle lobe of the cerebellum, and are due to its activity under the influence of impulses from the muscles, the semicircular canals, &c. On this hypothesis we can understand the origin of perceptions of posture and of movement which are not true sensations; active states of the cortical motor cells must correspond to the states of the muscles in passive as well as in active movement (see vol. i, p. 12). It is, moreover, noteworthy, in connection with the passage of the true sensory impulses by the fillet, that a connection of this with the middle lobe of the cerebellum (by fibres of the middle peduncle) has been lately described.†

The posterior third of the internal capsule contains not only the sensory path from the limbs, but also the optic path, and it also contains the paths for the other special senses; those for hearing and taste ascend to it from the pons in the tegmentum. How the path for smell reaches it is at present unknown. This is the "sensory crossway" of Charcot, in which a lesion causes hemianæsthesia, hemianopia, and loss of the other special senses, all on the side opposite to the lesion.

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* Senator, 'A. f. Psych.,' xi, p. 725, and xiv, p. 663; Goldscheider, 'Char. Ann.,' xvi, 1891, p. 162.

† Marchi, 'Riv. sper. di fren., &c.,' 1891.

Central Ganglia.—The corpus striatum and optic thalamus present considerable differences in their connections, and these probably indicate fundamental differences in function, although we are still almost entirely in the dark as to the nature of their function.

The grey matter of which the *optic thalamus* is composed consists of fine nerve-cells, among which narrow tracts of fibres pass. At the surface is a layer of white fibres, the "zonal stratum," and outside this again a very thin layer of gelatinous grey matter, continuous with that lining the third ventricle and surrounding the central canal of the cord. It is the union of this layer on the two thalami that constitutes the posterior commissure. The posterior extremity of the thalamus is distinguished as the "pulvinar," and between the extremities of the two thalami lie the anterior corpora quadrigemina. The grey matter of which the thalamus is composed is divided by a thin stratum of white fibres (internal medullary lamina) into an *external* and an *internal* nucleus, and the lamina divides anteriorly into two, which join the capsular layer, and with it enclose a small *anterior* nucleus. The most important connections of the thalamus are with the cortex, the optic tracts, and the tegmentum of the crus. It is connected with all parts of the cortex by fibres that pass from it to the internal capsule and corona radiata, of which they form a large part. They pass to all parts of the cortex; those from the pulvinar go to the occipital lobe, and constitute part of the "optic radiation" of Gratiolet. It is probable that most of these fibres conduct from the thalamus; nevertheless some fibres from the central convolutions to the thalamus have been found to degenerate downwards.* Fibres also pass between the thalamus and lenticular nucleus, but we do not know whether they end in the lenticular nucleus, or merely pass through it to the cortex.

The connection of the thalamus and optic tract will be described in the account of the latter. The chief downward connection of the thalamus is with the tegmentum. This may be said to end beneath the posterior part of the thalamus, in what has been termed the "subthalamic region," in which are certain collections of grey matter. Many fibres pass to the thalamus from the superior cerebellar peduncle, going through the red nucleus. The extent of the connection of the thalamus with the fillet and the reticular formation is a subject on which there is much difference of opinion. Wernicke finds the chief origin of the fillet in the thalamus, while Flechsig could trace no connection between them.

Corpus Striatum.—The caudate nucleus is more uniform in structure than is the lenticular nucleus. In the latter two narrow laminæ of white fibres, almost vertical in direction, divide the grey substance into three zones, inner, middle, and outer (Fig. 29). In consequence of the shape of the nucleus, the outer zone is the most extensive, both in the antero-posterior and vertical direction; and it is also darker in tint than the other parts. Each part of the corpus striatum consists of nerve-cells, large and small, and interlacing fibres. The fibres are far more numerous in the lenticular than in the caudate nucleus; but it is probable that many of these merely pass through the former, while the fibres that enter the caudate nucleus are connected with its cells. Moreover the outer part of the lenticular nucleus actually blends with the caudate nucleus in front by tracts of grey matter which pass through the internal capsule. To the outer side of the lenticular nucleus is the white layer of the "external capsule," and the grey lamina of the "claustrum." According to Kowalewsky* all three limbs of the lenticular nucleus receive fibres from the internal capsule and corona radiata, while the external capsular fibres are connected with some if not all those limbs. Further, all these limbs are connected with each other, and he regards the lenticular and caudate nuclei as together forming one ganglion, divided into two by the internal capsule.

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* Mannkopf, 'Zeitsch. f. kl. Med.,' 1884, Bd. vii, Sup.

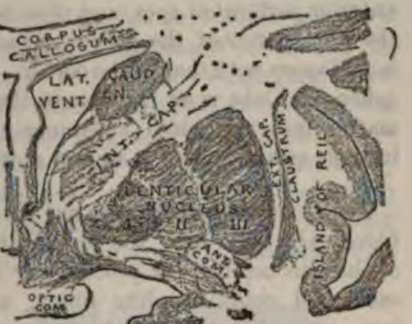

FIG. 30.—Diagram of a transverse section of the lenticular nucleus and internal capsule. I, II, III, indicate the three parts of the nucleus; ANT. COM., section of the bundle of fibres of the anterior commissure to the temporo-sphenoidal lobe.

It is doubtful whether the corpus striatum has any connection with the cortex, and the old hypothesis that its cells interrupt the fibres which conduct motor impulses seems to be altogether wrong. Meynert thought that many fibres pass from the caudate nucleus to the cortex; but the researches of Wernicke and others make this connection very doubtful. It is extremely difficult to ascertain whether the lenticular nucleus is connected with the cortex, on account of the number of fibres that pass through it and do not end in it. Fibres, however, pass from the caudate nucleus to the internal capsule, and others pass to the crus through the lenticular nucleus. These caudate fibres seem to end, as we have seen, in the pons, and to be connected, through the pontine grey matter, with the fibres of the middle cerebellar peduncles. The lenticular nucleus differs from the caudate nucleus in being extensively connected with the tegmentum of the crus cerebri by many fibres, and especially by those of the lenticular loop. It is also connected in a similar manner with the superior cerebellar peduncle. The connection of the corpus striatum with the cerebellum is thus very considerable. When the cerebellum is absent, the corpus striatum is reduced to a third of its normal size. Flechsig and Wernicke consider that it must be regarded as a central organ, analogous to the cortex. The latter has also pointed out that—the lenticular nucleus being continuous with the grey matter of the anterior perforated spot, and this being continuous with the cortex—the grey matter of the corpus striatum may even be conceived to be homologous with that of the cortex. The "amygdala" of the temporo-sphenoidal lobe is an instance of the development of the cortical grey matter into the white substance to such an extent as to appear, in some sections, as if it were a central mass.

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* 'K. Akad. der Wissensch.,' 1882.

The *Corpora quadrigemina* are masses of grey matter mingled with fibres, which lie over the aqueduct of Sylvius and the tegmentum of the crus cerebri. A process or "brachium" extends forward from each, and contains white fibres, which proceed from a superficial layer immediately beneath the convex surface. The process from the anterior tubercle sends fibres to the external geniculate body, and, by the posterior portion of the internal capsule, to the tegmental or optic radiation. That of the posterior tubercle goes to the internal geniculate body, and is thus connected, according to Von Gudden, with the commissural fibres of the optic tract, which at the optic chiasma turn back, along the opposite optic tract, to the opposite internal geniculate body and corpora quadrigemina.

The quadrigeminal bodies receive many fibres from the tegmentum, chiefly from the reticulate formation, and, according to some authorities, from the fillet. From the grey matter of the corpora quadrigemina fibres pass directly to the subjacent nucleus of the third nerves (Meynert).

We have no direct evidence of the function of these ganglia. The results of experiment are difficult to interpret, and these bodies appear to have a higher relative importance in animals. In man they are scarcely ever the seat of isolated disease. It is not probable that they are directly concerned in the function of vision. Stimulation of either the anterior or posterior causes dilatation of the pupils, first of that on the side opposite to the tubercle stimulated; while further stimulation causes tonic and tetanic spasm, ending in opisthotonos, and stimulation of the posterior causes the animal to cry out (Ferrier and others). The relation to the optic nerve and oculo-motor nuclei, and likewise some of the results of experiment, strongly suggest that these bodies are concerned in the adjustment of ocular movements to visual impressions. The spasm produced by their stimulation must be of reflex origin, and suggests important connections; but of the nature of these we have at present no evidence.

ORIGIN OF THE CRANIAL NERVES.

The surface attachment of the cranial nerves is too well known to need repetition here. Their relative position is shown in the accompanying figure (Fig. 31), and is of considerable importance because it determines the grouping of nerve palsies in diseases of the base of the brain. The nerves that arise nearest the middle line, and are therefore most readily affected on both sides by a single lesion, are the third nerves. The two sixth nerves are also near together, and so are the two hypoglossal nerves, but the latter are seldom both affected by disease outside the medulla because each passes outwards, and they are separated by the prominent anterior pyramids. The two sixth nerves suffer, on the other hand, with great frequency, because they have a long course, not far apart, over the most prominent part of the pons, and they are readily affected by distant pressure. The nerves that are farthest apart, and are least frequently damaged together by a basal lesion, are the two fifth nerves.

With regard to the relative position of the nerves, the fifth occupies a comparatively isolated position at its surface attachment: the sixth nerve is the nearest, and is most frequently associated with it in paralysis. As the fifth nerve enters the dura mater the third and fourth nerves are also near it (Fig. 32). The contiguity of the facial and auditory, and also that of the glosso-pharyngeal, vagus, and spinal accessory, are well known. It should be especially noted that the hypoglossal passes outwards close to the spinal accessory, and hence these two nerves not unfrequently suffer together, and palsy of the tongue is then conjoined with that of the vocal cord on the same side.

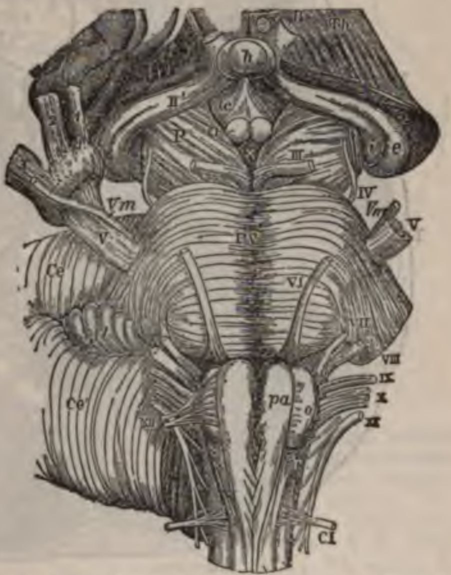

FIG. 31.—Origin of the cranial nerves (indicated by the roman numerals). Th, thalamus; t c, tuber cinereum; h, pituitary body ("hypophysis cerebri"); P, peduncle; P V, pons Varolii; a, corpora albicantia; Ce, cerebellum; pa, anterior pyramid; o, olivary body. (After Henle.)

The deep origin of these nerves (with the exception of the first two) is from a series of tracts of grey matter which are situated in front of the central cavity of the mesencephalon, and extend from the level of the anterior corpora quadrigemina above to that of the decussation of the pyramids below. Above and below, these tracts lie near the middle line, but beneath the floor of the fourth ventricle they extend almost from one side of the floor to the other. Most of the nuclei are situated between the reticular formation and the floor of the ventricle, but some lie on the outer side of the reticular formation.

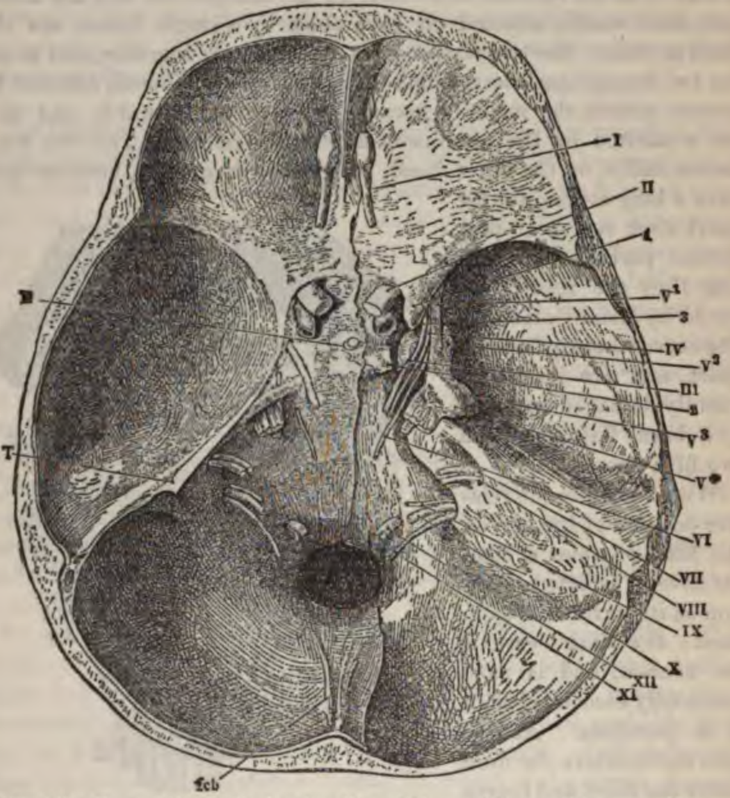

FIG. 32.—Base of the skull, showing the relative positions of the nerves (I—XII) as they enter the dura mater, which has been removed on the right side from the Gasserian ganglion, V*; f c b, cut edge of falx cerebelli; H, pedicle of pituitary body or hypophysis cerebri; T, cut edge of tentorium. (After Henle.)

It is probable that the upward path from those nuclei that are sensory in function lies in the reticular formation, while that from the brain to the motor nuclei runs chiefly with the pyramidal tracts, as we have already seen. The tracts of grey matter which constitute these nuclei are, for the most part, of small transverse sectional area, but of considerable length.

The *hypoglossal* nucleus (Figs. 28, 34, 35) is situated close to the middle line, and extends from just above the decussation of the pyramids to the calamus scriptorius of the fourth ventricle, at the point of which it lies beneath the prominence that adjoins the raphe. Below, it is situated in front of the central canal. The nerve-fibres pass from it through the inner part of the reticular formation and olivary body, and then curve outwards between the latter and the anterior pyramid. Besides the chief hypoglossal nucleus there is also the small-celled nucleus of Roller, *nucleus centralis*, which lies ventrally to the chief nucleus and surrounds its root-bundles. There are also some large cells lying among the roots of the nerve and probably reinforcing them, constituting the so-called *nucleus accessorius* of Duval. The nuclear origin of the fibres for the palate and vocal cords running in the spinal accessory nerve may possibly be in the lower part of the hypoglossal nucleus.[*]

Accessory, vagus, and glosso-pharyngeal.—A group of nerve-cells behind the hypoglossal nucleus, and behind the level of the canal, is the lower part of the nucleus of the *spinal accessory* nerve[†] (Fig. 28, *sp. a. nu.*), and from it the fibres course outwards (*Sp. A*) through the lateral part of the medulla. As the canal opens out into the fourth ventricle, this nucleus passes to the outer side of the hypoglossal, and the fibres pass forwards between the reticular formation and the restiform body. The nucleus is the lowest part of a tract of grey matter, the upper part of which lies beneath the floor (at the "ala cinerea") outside the eminentia teres, and gives origin, above the upper fibres of the spinal accessory, to the fibres of the pneumogastric, and then to those of the glosso-pharyngeal. The filaments of origin of these nerves form a continuous series, and it is scarcely possible to say where one ends and another begins. They all have a similar course to the surface, emerging beside the prominence of the resti-

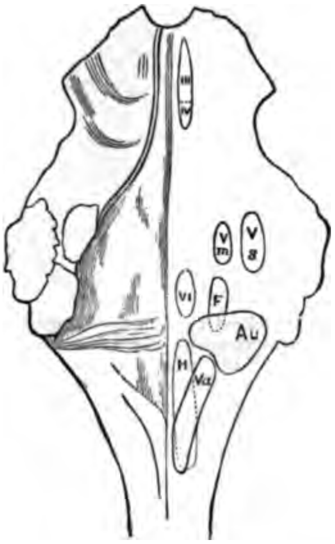

FIG. 38.—Diagram of the relative position of the nerve nuclei beneath the floor of the fourth ventricle. III, third nerve nucleus; IV, fourth; V s, middle sensory nucleus of the fifth; V m, motor nucleus of fifth; VI, sixth; F, facial; Au, auditory; H, hypoglossal; V a, vago-accessorial nucleus, the upper part giving origin to the pneumogastric, the lower to the highest fibres of the spinal accessory. Where one nucleus lies beneath another its outline is indicated by a dotted line.

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[*] Lockhart Clarke, 'Phil. Trans.,' 1868.

[†] The lower fibres of the nerve arise, not from the spinal accessory nucleus, but from the anterior cornu of the upper cervical cord. These fibres supply the muscles of the neck.

form body, and all pass through a group of longitudinal fibres, more or less oval in transverse section, the ascending root of the fifth nerve (Fig. 34, V asc). No other nerve-roots pass through this bundle. It may be noted that the fibres of the spinal accessory which arise from the medulla are those that innervate the muscles of the larynx, and are thus associated in origin with the fibres of the great respiratory nerve, the vagus.

FIG. 34.

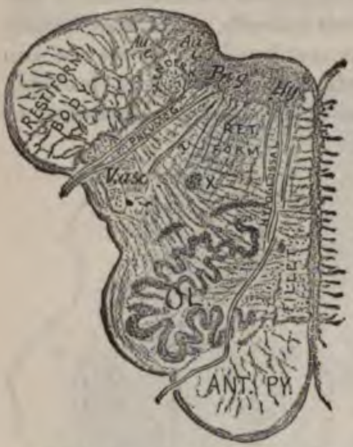

FIG. 35.

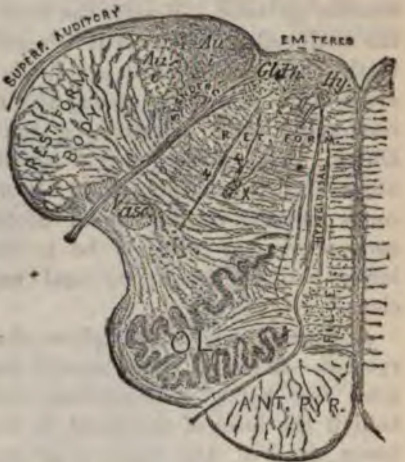

FIG. 34.—Origin of pneumogastric. Hy, hypoglossal nucleus; Png, chief nucleus of pneumogastric; Au, i e, lowest part of internal and (so-called) external auditory nuclei; V asc, ascending root of fifth; X, nucleus of unknown function in front of reticular formation (RET. FORM.); *x*, fibres passing forwards from the neighbourhood of the vagal nucleus.

FIG. 35.—Diagram of half-section of pons at the level of the glosso-pharyngeal nucleus, *Gl.Ph.*; *Hy*, hypoglossal nucleus. The other lettering is the same as in Fig. 34.

Moreover, in this relation, another connection of these nerves is important. To the outer side of these nuclei, and of the fibres proceeding from them, is another bundle of longitudinal fibres, rounded in section and small in size. It is the "*slender column*" of Lockhart Clarke (Fig. 34). It extends upwards as high as the highest part of the glosso-pharyngeal nucleus, while downwards it passes into the deep part of the lateral column of the cord; its fibres have been traced as far as the middle of the cervical enlargement, and may extend lower still. Some fibres of the glosso-pharyngeal, pneumogastric, and spinal accessory nerves pass into it, or rather arise from it, and hence it has been termed their ascending root, or the ascending root of the glosso-pharyngeal.* It is supposed to be connected with the process of respiration, to which the spinal accessory and vagus have such important relations. Hence Krause termed it the

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* "Common ascending root of the lateral mixed system" by Meynert.

"respiratory column." Division of it on both sides is said to arrest all movements of respiration.*

The nucleus of the *pneumogastric* and *glosso-pharyngeal* nerves is thus a tract of grey matter which lies beneath the outer half of the floor of the fourth ventricle, having the hypoglossal nucleus on the inner side, and, on the outer side, the restiform body below (where the pneumogastric arises, Fig. 34), and the lower part of the auditory nuclei above (where it gives origin to the glosso-pharyngeal, Fig. 35). Some fibres arise from groups of large nerve-cells in the deeper part of this tract, and these are probably the motor fibres of the nerves; such cells are especially conspicuous in the glosso-pharyngeal portion (Fig. 35). Other fibres arise from smaller nerve-cells in the more superficial grey part of the nucleus. Some fibres seem to come from the "slender column," as already stated, and others have been traced inwards to the raphe. According to Meynert, some fibres of the pneumogastric pass to the cerebellum; the gastric functions of the nerve are readily deranged by disturbance of the equilibration, as in the vomiting of vertigo. Lastly, from the neighbourhood of this nucleus, where the nerve enters it, fibres pass forward towards the deep part of the tegmentum (*x x*, Figs. 34 and 35). These have been thought to arise from a small collection of large nerve-cells (X), but most of them certainly pass by this nucleus† to nerve-cells near the surface.

The deep origin of the *auditory* nerve is still involved in some uncertainty. Its attachment to the medulla (at the junction of this with the pons) is by two roots, one of which (Fig. 37) winds round the restiform body (inferior cerebellar peduncle), while the other (Fig. 36) passes into the substance of the medulla. The former is termed the superficial (or outer) root, and the latter the deep (or inner) root. The former is a little posterior to the latter, so that the two are not shown in the same section; the lowest superficial fibres are on a level with the glosso-pharyngeal nucleus (Fig. 35), but the nuclei of the auditory nerve extend still lower in the medulla, as far as the middle of the vagal nucleus (Fig. 34). The outer or superficial root contains fibres, continuous with those of the cochlear part of the auditory nerve, and, in coursing round the medulla, contains some grey matter mingled with its fibres. Some of the fibres are continuous with those of the auditory striæ, which seem to pass to the middle line and opposite side of the medulla.* Many of the fibres end in a thick tract of grey matter, which occupies the inner two thirds of the floor of the fourth ventricle at this level, the "inner auditory nucleus" (*Au. i*, Figs. 34–37), which is commonly known as the chief nucleus of the nerve. Outside this is a smaller area containing many groups of longitudinal fibres, which is commonly known as the "external auditory nucleus" (*Au. e*), or Deiter's nucleus (see foot-note); to it some fibres of the superficial root seem to go, but it is probable that they merely pass through it, and that its chief connection is with the inner or deep root.† As the nerve courses round the medulla many fibres curve inwards into the restiform body; some may change their direction and join the restiform fibres, but others appear to pass transversely through the restiform body, and to be continuous with the transverse fibres of the tegmental portion of the pons. What their destination is we do not know. Meynert thinks that they may pass to the opposite restiform body and the cerebellum.

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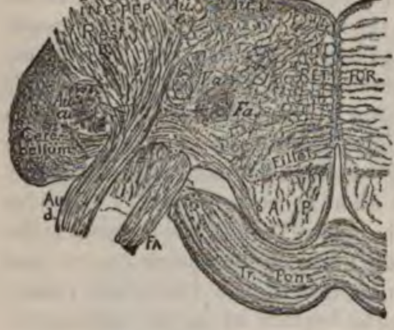
* Gierke. At the same time the division of this tract without injury to adjacent structures, on a living animal, is manifestly impossible. The connection with the vagus has been described by almost all investigators, but has been contested by Spitzka ('New York Med. Record,' 1884).

† The nucleus has been termed the "anterior nucleus" of these nerves by some, by others the "nucleus ambiguus." It is not certain that the fibres that go towards it are root-fibres. I believe they are connected with scattered nerve-cells which lie among the arciform fibres, between the ascending root of the fifth, and the olivary body. I have many times traced the fibres to this part, and observed that these cells send a process in the direction of these fibres. The nucleus X is not, as might be imagined from its position, continuous with that of the facial nerve.

FIG. 36.



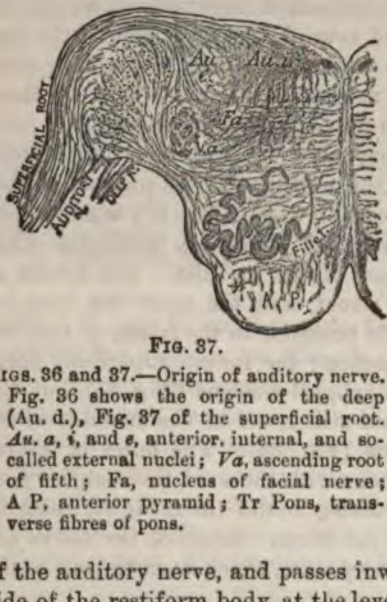


FIG. 37.

FIGS. 36 and 37.—Origin of auditory nerve. Fig. 36 shows the origin of the deep (Au. d.), Fig. 37 of the superficial root. *Au. a*, *i*, and *e*, anterior, internal, and so-called external nuclei; *Va*, ascending root of fifth; Fa, nucleus of facial nerve; A P, anterior pyramid; Tr Pons, transverse fibres of pons.

The deep root (Fig. 36) is continuous with the vestibular part of the auditory nerve, and passes inwards and upwards, on the inner side of the restiform body, at the level of the lowest fibres of the pons. It goes to the external auditory nucleus above described. But many fibres curve outwards into the restiform body and pass towards the middle lobe of the cerebellum. These are probably the fibres that come from the semicircular canals, and subserve, not auditory sensations, but impressions from the canals determined by the position and movement of the body. A few fibres may reach the "internal auditory nucleus," while others turn outwards before reaching the restiform body to an important collection of grey matter which lies in front of the latter, the "anterior or accessory auditory nucleus" (*Au a*, Fig. 36). Lower down, this nucleus lies in the angle between the two roots of the nerve, and (together with the cells in the outer division of the nerve) seems to give rise to a considerable number of the fibres of the nerve.*

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* Their connection with the nerve is doubted by Bechterew. See 'Neur. Cent.,' May, 1892.

† Monakow found it atrophied after hemisection of the lower part of the medulla ('Arch. f. Psych.,' Bd. xiv). See also, on this subject, Onafrowicz (ib., Bd. xvi). This nucleus has been also termed "Deiter's nucleus," as an indifferent designation, but the name has not come into general use, and it is marked *Au. e* in the diagrams, in accordance with prevailing usage.

Of the auditory path to the hemisphere we know only that it passes by the posterior part of the internal capsule. It probably passes in the most superficial layer of the tegmentum of the crus.† Meynert thinks that it passes through the cerebellum, and not through the pons; and, improbable as this may seem, it cannot be said that we have any facts at present inconsistent with the hypothesis. The central connections of the accessory nucleus are numerous, and include one with the cerebellum; their meaning is too uncertain to make it worth while to describe them.‡ The chief connection is probably with the tegmental region, possibly with the opposite flocculus. The functions of the auditory nerve are very closely connected with those of other cranial nerves, more so indeed than we can readily perceive, and the connections are of great physiological importance.

Above the auditory striæ the most prominent part of the eminentia teres marks the position of the nucleus of the *sixth nerve* (Fig. 38), to which the fibres pass through the inner part of the reticular formation. They enter the nucleus on its inner side. The fibres of the *facial nerve* pass outside the reticular formation to the neighbourhood of the sixth nucleus, where they form a loop and turn downwards, most of them in a compact bundle around the upper extremity of the nucleus, but some through the nucleus itself. They then radiate downwards, forwards, and outwards to a column of nerve-cells (Fa, Figs. 36, 37), which lies above the outer part of the fillet, near the grey tract known as the "superior olivary body," supposed to be related to the conjugate rotation of the head and eyes, an opinion supported by the entrance of fibres into it from the sixth nucleus. There is also a very close physiological connection between the muscles of the lip and tongue, and the facial nucleus extends down almost to the level of the hypoglossal nucleus, although the two are some distance apart, and we do not know whether the connection between the lips and tongue is subserved by fibres that pass between the two nuclei, or whether (as is possible) the nerve-fibres for the lips actually arise from the hypoglossal nucleus. Many of the fibres of the facial nerve, turning downwards at the loop, have a longitudinal course in the inner part of the reticular formation, and may readily reach the hypoglossal nucleus.*

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* So that it has been regarded as the chief auditory nucleus, although by some it is looked upon as analogous to a spinal ganglion. But Bechterew traces many fibres of this root to the grey matter in the lateral wall of the fourth ventricle—to what has been termed the "angular nucleus," and has also been named after him ('Neur. Cent.,' 1887).

† See a case published by me, 'Lancet,' March 15th, 1879, in which bilateral deafness was caused by a tumour of the corpora quadrigemina, damaging the superficial layer of the tegmentum on each side.

‡ See Baginsky, 'Neur. Cent.,' 1889; Flechsig, ib., 1890.

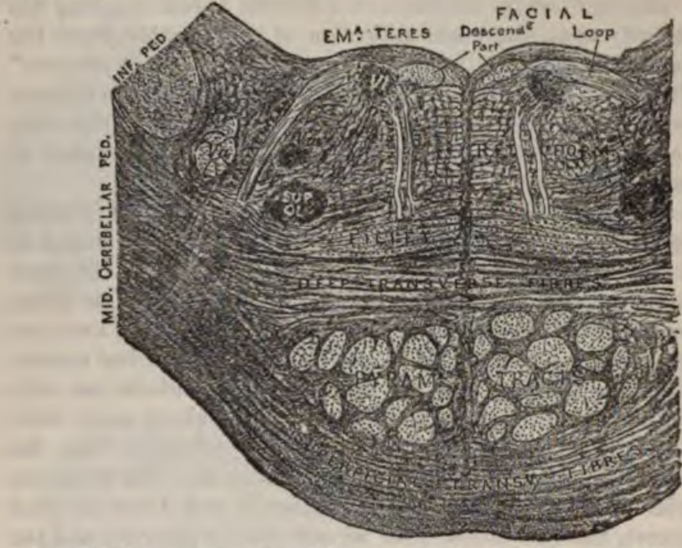

Fig. 38.—Transverse section of the pons at the level of the nuclei of the sixth nerves (VI); RET. FORM., reticular formation; Vm, motor nucleus of fifth nerve; *Va*, its ascending root seen in section; SUP. OL., superior olivary body.

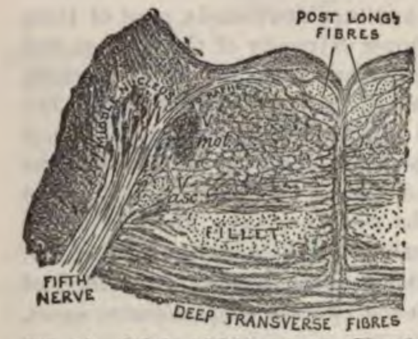

Fig. 39.—Origin of fifth nerves. *V mot.*, motor nucleus; *V asc.*, ascending root.

Similarly it has been suggested that the fibres in the facial supplying the orbicularis palpebrarum, frontalis, and corrugator supercilii are derived from the nucleus of the third nerve, and reach the facial by way of the posterior longitudinal bundles.† But for evidence of these hypotheses we have still to wait.

The origin of the *fifth* nerve (Fig. 39) is very extensive, its deep connection reaching from the level of the anterior quadrigeminal tubercle to the lowest part of the medulla. This is not surprising when we reflect that this nerve represents the sensory roots of all the motor cranial nerves, from the third to the hypoglossal. The two parts of the nerve pass backwards and inwards through the outer part of the pons, or rather through the inner part of the middle peduncle of the cerebellum. The smaller, motor root is a little higher up than the other. The sensory root reaches the outer part of the tegmentum, beneath the outer edge of the floor of the fourth ventricle, and there some of its fibres terminate in small collections of grey matter, which are collectively termed the *middle nucleus*, or "chief nucleus." It is certainly not the chief origin of the nerve, for by far the largest number of the fibres turn downwards and descend the pons and medulla, as a compact collection of bundles, lying in the same relative position, on the outer side of the tegmentum. This is termed the ***ascending root***, but, to avoid ambiguity, may be better called the *lower root*. It is a conspicuous object in all sections, and is shown in most of the preceding figures (*V asc.*). It becomes smaller as it passes down the medulla, but may be traced to the upper cervical region, and the source of the lower fibres is uncertain. It may be from nerve-cells of the posterior horn (Bechterew). Some fibres of the nerve course inwards, beneath the floor of the fourth ventricle; passing through the posterior horizontal fibres, they reach the raphe, and probably go to the opposite half of the pons: their precise origin is not known. This is termed the *medial root*. It is easily mistaken for the loop of the facial nerve, which has a similar position a little lower down the pons. Fibres probably connect the two motor nuclei, and others may bring the motor path from the other side of the pons (and other hemisphere). A few fibres are said to turn outwards to the cerebellum, but this connection, probable as it is, still needs confirmation. The ***descending root*** of the nerve (better termed the ***upper root***) consists of a series of bundles which pass upwards* and lie outside the aqueduct of Sylvius, where they are arranged somewhat in the form of a crescent (Figs. 40 and 41).† They can be traced as far as the upper part of the corpora quadrigemina, but lessening in number, and they apparently rise from large rounded nerve-cells that lie adjacent to the root. The root has commonly been regarded as one origin of the sensory portion of the nerve, but it has been stated that most of its fibres enter the motor division.‡ Almost all the fibres of the ***motor root*** end in a large collection of nerve-cells (*V mot.*), which lies on the inner side of the sensory root, in the outer part of the tegmentum, and extends for a short distance down the pons.*

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* The closeness of the central relation between the lips and tongue is shown both by their simultaneous affection in disease, such as labio-glossal paralysis, and by the fact that the transverse fibres of the tongue and the orbicularis oris can only contract together.

† See Mendel, 'Neur. Cent.,' 1887, and Tooth and Turner, 'Brain,' 1891.

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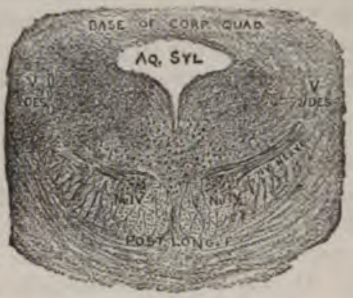
* The terms ascending and descending have been adopted apparently under the influence of the word "root," but the fibres conduct in the direction opposite to that indicated by the names. Hence the alteration I have suggested.

† See also Fig. 24 (p. 30), in which the crescent on each side of the aqueduct represents the descending root.

‡ Henle, Forel, Bechterew, &c. In spite of the high character of these authorities, the statement is so opposed to the opinion of other investigators that the point must be regarded as at present unsettled. Both coarse and fine fibres are said by Obersteiner to proceed from the same cells, the latter passing upwards. See, in connection with this point, "Facial hemiatrophy," at a subsequent page.

Each *fourth nerve*, arising from the valve of Vieussens (superior medullary velum), decussates within the substance of the valve with its fellow (Fig. 41). It is the only nerve that decussates between its surface origin and nucleus, with the exception of the optic. In its course in the velum the nerve divides into a series of bundles, which curve forwards and upwards, round the central grey matter lining the aqueduct of Sylvius, to a collection of nerve-cells at the hinder and outer part of the posterior longitudinal fibres (Fig. 40). The nucleus lies beneath the junction of the anterior and posterior quadrigeminal bodies and beneath the latter, but it is not sharply defined from the third nucleus, so that many of the fibres pass the whole length of the nates before reaching the nucleus. Hence, according to the position of the section, the bundles of origin are divided transversely, obliquely, or longitudinally (see Figs. 40 and 41). Some fibres seem to pass to

FIG. 40.



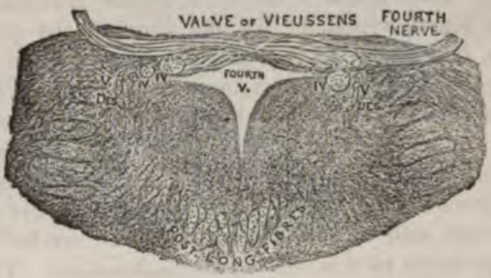


FIG. 41.

FIGS. 40, nucleus, and 41, origin of fourth nerves. V. DES., descending root of the fifth nerve; IV, IV, sections of fasciculi of the fourth nerves.

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* A connection with the l. cœruleus (s. ferruginea) has been asserted and also denied (Bechterew).

the posterior longitudinal bundles; some (Westphal) to a group of round dark cells behind these, near the raphe.*

The fibres of the *third* nerve arise from a column of nerve-cells that occupies the anterior part of the central grey matter below the aqueduct of Sylvius, beneath the anterior quadrigeminal bodies and the posterior commissure (Fig. 24, p. 30). The cells from which the fourth nerve arises are in the lowest part of this column. The fibres of origin of the third nerve pass forwards, curving outwards through the red nucleus, and then inwards, to the side of the crus, where they emerge. The cells lie in a double series: a smaller, towards the dorsal aspect, gives origin to fibres that cross the middle line; and a larger "ventralwards," from which the fibres pass through the posterior horizontal bundles which, as we have seen (p. 37), are connected with this nucleus, and seem to establish an association between it and the sixth nucleus, through which the external and internal recti act together.† The nucleus of the third nerve consists of a series of groups, almost fused, corresponding to the several functions subserved by the nerve, the most anterior being related to accommodation, the next to the reflex action of the iris, while the remainder of the nucleus, comprising its chief part, subserves the external ocular muscles in the following order, from before backwards:—Internal rectus, superior rectus, levator palpebræ superioris, inferior rectus, inferior oblique. It is highly probable that the same arrangement obtains in man, and that the serial disposition of the filaments of origin of the nerve corresponds to the series of functions and of centres. But the contiguity of these is lateral as well as antero-posterior, and the disposition thus complex (see "Motor Nerves of the Eyeball"). A complex arrangement also seems to subserve the association of the internal and external recti. When acting together both these muscles are innervated through the nucleus of the sixth, disease of which causes loss of at least the associated action of the internal rectus. The path from the sixth nucleus to the origin of the internal rectus is doubtless by the posterior longitudinal fibres; and there is some reason to regard the superior olivary body as concerned in the action. The subject is considered more fully in the account of diseases of the oculo-motor nerves. Fibres pass to the third nuclei from the grey matter of the quadrigeminal bodies, and doubtless subserve the relation between visual impressions and ocular movements. The downward path from the cortex in the internal capsule seems to be at or in front of the angle or just behind it, according as a section divides their oblique course. (See Beevor and Horsley, 'Phil. Trans.,' 1890.)

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[*] J. Stilling describes a fine root from the cerebellum which runs through the lingula and joins the trochlear, perhaps without crossing. It is supposed that the connections of the trochlear with the cerebrum, testes, and posterior longitudinal bundle are the same as those of the oculo-motor, to be afterwards described.

[†] The upper third of the nucleus has an oval section, and consists of large pale cells (Perlia, 'A. f. O.,' 1890, who describes a minute subdivision of the nucleus).

The origin of the *optic nerves* is complex and still imperfectly understood. At the optic chiasma a partial decussation takes place, the fibres from the nasal half of each retina cross, and as the temporal half of each field of vision (subserved by the nasal half of the retina) is (functionally) the larger,* more fibres cross than pass uncrossed. Thus each optic tract contains the fibres from the same-named half of each retina, which conduct impressions from the other-named half of each field. Fibres also pass from one tract to the other by the posterior portion of the chiasma. The old opinion that fibres pass from one optic nerve to the other by the anterior part of the chiasma seems to be erroneous, but some go to the central grey matter lining the third ventricle.† In the nerve, the uncrossing fibres lie in thick bundles in the lateral (outer) part. In the tract, the fibres from the opposite eye lie chiefly on the inferior (basal) surface; the direct fibres lie in the superior part of the tract; and the fibres from the opposite tract are on the inner side (v. Gudden). The fibres from the lower half of each retina lie outside to those from the upper half.‡ Beneath the posterior extremity of the thalamus the optic tract divides into two parts. The outer is the larger, and goes to the optic thalamus, external corpus geniculatum, and anterior quadrigeminal body. All these structures atrophy after extirpation of the eye of young animals (v. Gudden). From each of these bodies fibres pass to the posterior (sensory) portion of the internal capsule, and thence to the white substance of the occipital lobes, in the "optic radiation" of Gratiolet. Some fibres of the tract have been said to pass directly to the hemisphere, but it is doubtful whether any have this direct course. Of these intermediate stations, the external corpus geniculatum has been commonly regarded as that which is of chief importance in connection with the visual fibres, since its atrophy has been frequently observed in cases of long-standing atrophy of the tract. But many recent observations establish the fact that disease limited to the posterior extremity of the optic thalamus may cause hemianopia, and it seems doubtful whether the symptom is caused by disease of the external geniculate body.§ Hence we must regard the pulvinar as the intermediate visual centre, and the precise function of the external corpus geniculatum becomes again mysterious. The inner root of the optic nerve goes to the internal geniculate body, and is said to contain only those fibres which, at the optic commissure, turn back to the opposite optic tract (v. Gudden). Through this body the nerve also appears to be connected with the posterior corpus

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* See "Optic Nerve."

† Obersteiner.

‡ Marchand, 'A. f. O.,' xxviii, and Magnus, ib. See, however, also Delbrück, 'A. f. Psych.,' xxi, 746.

§ It is commonly believed that the fibres are interrupted by nerve-cells in these ganglia, but Darkschewitsch ('Arch. f. Anat. u. Phys.,' 1886) has asserted that they pass through without interruption, a fact that can scarcely be proved by the method of investigation employed by him. Secondary degeneration from a lesion of the occipital lobe causing hemianopia has been traced into the pulvinar, but no further (Richter, 'Arch. f. Psych.,' xvi, 638).

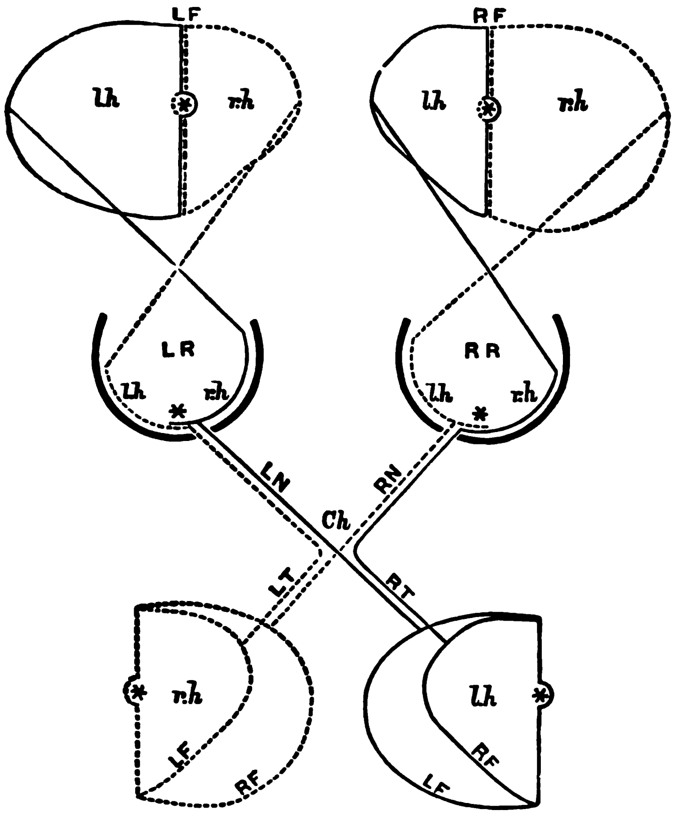

Fig. 42.—Diagram of the relation of the fields of vision, retina, and optic tracts. R F, L F, right and left fields—the asterisk is at the fixation-point; R R, L R, right and left retina—the asterisk is at the macula lutea; *l h*, *r h*, left and right half of each retina, receiving rays from the opposite halves of the fields; R N, L N, right and left optic nerves; *Ch*, chiasma; R T, L T, right and left optic tracts; below are the superimposed halves of the fields from which impressions pass by each optic tract.

quadrigeminum, but some fibres seem also to reach the anterior body.[*] The connection of the optic nerves with these bodies probably subserves the relation between visual impressions and ocular movements. The relation of the parts concerned in vision is illustrated and also confirmed by an instructive case of congenital atrophy of one occipital lobe, extending as far as the middle of the posterior limb of the fissure of Sylvius. There was also atrophy of the pulvinar, the optic tract, the external geniculate body, the superficial layer of the anterior quadrigeminal body, and its anterior brachium.* The relation of the cortex to vision has been already mentioned, and some other points in connection with the function of the nerves will be described in the account of their diseases.

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[*] Stilling, Obersteiner.

The central connections of the *olfactory nerve* are still but little known. The external root, crossing the fissure of Sylvius, passes to the temporal lobe, towards the anterior part of the uncinate convolution. There is some evidence, as we have seen, that an olfactory centre exists in this locality. Some fibres (according to Obersteiner) pass to the optic thalamus on the same side. The fibres of the inner root go to the anterior commissure, one part of which, in animals, is large in proportion to the size of the olfactory nerves, and is regarded as commissural between the two olfactory bulbs. But there is evidence that the path from one olfactory nerve reaches the cortex of the opposite hemisphere, since the sense of smell has been affected, together with the other special senses on the same side, by disease of the posterior part of the opposite internal capsule, and also by disease of the cortex (see p. 20). It is possible, as Charcot suggests, that the path damaged by disease involving the internal capsule is that which crosses by the anterior commissure.†

CEREBELLUM.

The division of the cerebellum into two hemispheres and a middle lobe, founded on external conformation, is borne out by the little we know of its function. Nevertheless all parts consist of similar structural elements. Branching folia everywhere constitute the superficial layer; the grey substance which envelops these folia, and, following all the involutions of their surface, is of enormous superficial extent, has an identical three-layered structure in all parts. Below a superficial "gelatinous stratum" is a layer that contains nuclei and branching fibres, which are the processes of the large "cells of Purkinje." These cells lie at the junction of this and the deepest layer of the cortex; the latter is composed of nuclear bodies similar in aspect to those that constitute the granule layers of the retina. The grey matter is not confined to the cortex. It exists also in the white substance as a series of masses or "nuclei." Of these the corpus dentatum lies in the inner part of the hemisphere, and closely resembles the olivary body of the medulla. In the white substance of the middle lobe is the "roof nucleus" (RN, Fig. 43), so called by Stilling because it lies just above the roof of the fourth ventricle. At the junction of the hemisphere and middle lobe are two other grey bodies, the "spherical" and "emboliform" nuclei (Stilling). The latter is the nearer to the dentate body.

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* Kreusner, 'Allg. Zeitschr. f. Psych.,' Bd. xlviii.

† The subject is involved in great obscurity. The other fibres of the anterior commissure pass into the temporal lobe, but they have not been traced to the uncinate gyrus. Meynert says that fibres pass back from the anterior commissure, and thus the olfactory path (whether from the nerve by the one part of the commissure, or from the opposite uncinate centre by the other part of the commissure) may reach, as it certainly does, the internal capsule, but Ganser denies that fibres pass backwards from the anterior commissure. As a further instance of the uncertainty as to the precise relations of the anterior commissure, most of its fibres have been found secondarily degenerated in a case of bilateral softening of the region of the lingual gyrus by Popow, who regards it as a commissure between these convolutions ('Wratsch,' 1886, and 'Cent. f. Nervenh.,' 1886, p. 684).

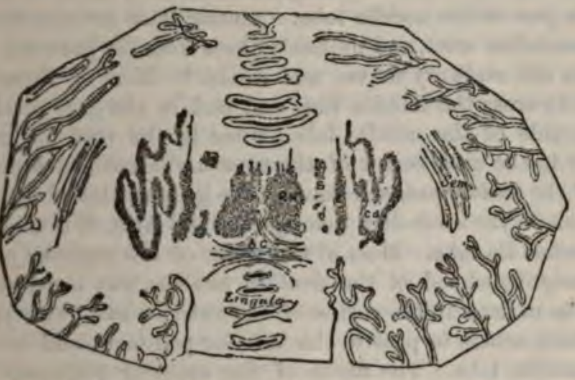

FIG. 43.—Nuclei of the cerebellum. C d, corpus dentatum; E, emboliform nucleus; S *s*, parts of the spherical nucleus; A C, anterior commissure; *Sem.*, semilunar tract.

The course of the fibres in the white substance is still imperfectly known. The difficulty of tracing them is extreme, unaided as it is by secondary degeneration, and the conclusions reached by different investigators do not altogether correspond. Although much has been written on the subject, the facts are so meagre, and their application is so uncertain, that a lengthy discussion of the subject is unprofitable. There are few well-marked tracts of fibres, and of those that can be distinguished, such as the semilunar tract (*Sem.*, Fig. 43) in the outer part of the hemisphere, the connections are doubtful. Some fibres of the hemisphere appear to pass in various directions between the different folia of the cortex; others pass inwards, or towards the middle line. Of the latter, some go to the dentate nucleus, and interlace around it in a felty capsule, the "fleece" of Stilling; others pass to the middle peduncle, and end in the grey matter of the pons, and through this are connected with the cortex of the cerebral hemispheres and with the corpus striatum, as we have already seen (p. 32). Other fibres pass to the middle lobe, part of which may have a commissural function. Flechsig states that many fibres connect the cortex of the superior vermiform process with that of the cerebellar hemispheres.

Of the fibres of the *inferior peduncle* many course to the neighbourhood of the dentate nucleus; some enter this nucleus, and apparently connect it with the olivary body. The destination of those that pass by the dentate nucleus is very uncertain. Stilling thinks that most go to the cortex of the hemisphere, and a few directly to the superior peduncle. Another and very important series of fibres of the inferior peduncle pass to the middle lobe. Among these are the fibres of the direct cerebellar tract, and probably fibres from the posterior pyramids and from the auditory nerves and nuclei.* Most of these fibres are believed to cross the middle line, and end in the grey matter of the opposite side of the middle lobe; some in the roof nucleus, others probably in the grey cortex of the superior vermiform process. The fibres of the *middle peduncle* connect the grey matter of the pons with the cortex of the cerebellar hemisphere, and also, it is said, the fillet with the middle lobe. Most of the fibres of the *superior peduncle* go to the neighbourhood of the dentate nucleus, but some are said by Stilling to radiate to the cortex of the anterior and outer part of the hemisphere, others to pass to the inferior peduncle, and others again to the middle lobe. The fibres of the superior peduncle cross the middle line beneath the corpora quadrigemina, and pass through the opposite red nucleus, but one group is said to ascend to the optic thalamus of the same side (Marchi).

The precise functions subserved by the cerebellum have been long a mystery. There is, however, abundant evidence, experimental and pathological, to show that this part of the brain is in some way connected with the co-ordination of movement, and especially with those muscular actions which maintain the equilibrium of the body. It appears, however, that this function is confined to the middle lobe. As Nothnagel first showed, the function is not impaired by disease of the hemispheres unless such disease compresses the middle lobe. We do not know to what extent this function is subserved by the roof nucleus, or by the grey matter of the middle cortex. We may reasonably assume that the fibres which go to the middle lobe from the cord, those of the direct cerebellar tract and (probably) those from the post-pyramidal nuclei, are connected with this function, since we have seen reason (in the account of the spinal cord) to believe that these fibres constitute the path of centripetal impressions from the muscles, interruption of which causes incoordination. All the tracts certainly or probably connecting the spinal cord with the cerebellum in man conduct upwards. The existence of connections between the cerebellum and other structures is no proof of downward conduction of impulses from it.

With this function, and with the relation of the upbearing paths from the muscles, we must associate the fibres which pass from the auditory nerves or nuclei to the middle lobe, since that part of the auditory nerve which comes from the semicircular canals conducts impressions determined by the position and movement of the body (acting on the endolymph), and disturbance of these fibres deranges the maintenance of equilibrium. The only effect of electrical stimulation of the middle lobe obtained by Ferrier was a movement of the eyes. It is not probable that there are centres for the ocular movements in the cerebellum, but a connection between the centres for these movements and the middle lobe is highly probable, since the relation of seen objects to the body is estimated unconsciously from the position of the eyes, and thus the degree of innervation of the ocular muscles must influence the mechanism for maintaining equilibrium. It is to be noted that most of these impressions, *e.g.* from the muscles of the legs and from the semicircular canals, do not influence consciousness directly, and so cannot, strictly speaking, be called "sensory." It may be that in the middle lobe of the cerebellum we have a mechanism by which the various centripetal impulses are combined and harmonised, and that an influence is thence exerted on the motor centres in the cerebral hemispheres, from which the muscles are excited to the necessary contraction. There is no actual evidence that the cerebellum aids co-ordination by direct downward influence on the spinal cord. On the contrary, such a connection is opposed to what we know of the voluntary motor process. Movements are arranged by the establishment of a certain relation between the activity of the motor cells of the cerebral cortex, and the influences which determine this relation must be exerted on those cells. Besides the true sensory impulses, we can conceive that an influence proceeds from the cerebellum, arising in it from the combined effect of the impressions from the muscles, from the semicircular canals, and from the centres which determine the position of the head and eyes (possibly the superior olivary body), and that this influence induces such a corresponding related activity of the motor cells of the cortex. We can also understand that the state of these cells thus induced acts upon consciousness, and thus gives rise to the perceptions of posture which are not true sensations.

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* In some of the lower animals some of the fibres of the direct cerebellar tract are said to degenerate downwards after extirpation of the cerebellum, but the existence of such fibres in man has not been ascertained (Marchi, 'Riv. sper. di fr.,' xvii).

The function of the hemispheres of the cerebellum is still mysterious. They lessen in size as we descend the scale of animals, until they disappear in birds, in which the whole cerebellum corresponds to the middle lobe of man. They are connected chiefly with those parts of the cortex of the cerebrum which chiefly subserve psychical processes. With these parts, moreover, the cerebellar hemispheres have this in common, that simple loss of substance causes no definite and recognisable loss of any function of the brain. The loss can apparently be compensated by other parts. Hence it seems possible that the old theory may be correct which assumes that the cerebellar hemispheres are in some way connected with psychical processes.*

* Extirpation of the whole cerebellum in an animal renders it dependent on vision

BLOOD-VESSELS OF THE BRAIN.

The blood-supply to the brain is a subject of great importance, because many diseases are due to the rupture or obstruction of vessels, and the distribution of the vessels (especially that of the arteries) explains the incidence of a large number of cerebral lesions.

DISTRIBUTION OF THE CEREBRAL ARTERIES.*—The blood-supply to the brain comes from the carotid and vertebral arteries. The mode of origin of the two carotids presents an important difference, which explains why embolism is more frequent on the left side than on the right. The left carotid arises directly from the highest part of the arch of the aorta, and its course is thus almost a direct continuation of the direction of the current of blood in the aorta, whereas the right carotid comes from the innominate, which arises from the aorta at an angle with the course of the aortic blood. Hence clots, washed from the cardiac valves, pass into the left carotid more readily than into the right.

There is a similar difference in the mode of origin of the two vertebrals. The left, usually the larger, arises from the left subclavian in the ascending part of its course, while the right subclavian gives origin to its vertebral when horizontal. Hence a plug can enter the left vertebral more readily than the right, because the direction of this vessel is that of the blood-current, and this is probably also the reason why the left vertebral is usually larger than the right. But this does not determine any difference in the frequency of embolic obstruction in the two posterior cerebral arteries, because, to reach these, the plug has to pass through the common basilar.

The "circle of Willis," it will be remembered, is formed by the two posterior communicating arteries, which pass, one on each side, from the internal carotids to the posterior cerebrals into which the basilar divides, and by the anterior communicating artery, between the two anterior cerebrals of the internal carotid. The usual arrangement is subject to occasional variations, which are important because they explain some of the anomalous facts of disease. The vertebrals often present abnormalities which will be described further on. One posterior communicating artery is sometimes very small, and occasionally one, usually the right, is abnormally large, and through such a vessel the posterior cerebral may even arise from the carotid, a minute branch from the basilar representing the usual origin of the vessel. Duret has even seen the chief blood-supply to the basilar come from the carotid by a large artery which entered the basilar in the middle of its course, the vertebrals and the lower part of the basilar being very small. Occasionally one anterior cerebral, almost always the right (Duret), is very small at its origin, and the anterior communicating artery is abnormally large, so that the right anterior cerebral virtually arises from the left. It is to be noted further that there are usually communicating branches between the posterior cerebrals and the anterior cerebellar arteries, which, with the posterior communicating arteries, constitute an important connection between the carotid and the basilar systems.

The internal carotid, on each side, divides into the anterior and middle cere-

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for the imperfect co-ordination which remains, and if the eyes are covered with a bandage all voluntary movement is at once abolished, and the creature seems to pass into a state of stupor (Borgherini, 'Neurolog. Centralbl.,' 1891, p. 649).

* Our knowledge of the distribution of the vessels within the substance of the brain is largely due to the researches of Duret, 'Arch. de Physiologie, 1873 and 1874; and Heubner, 'Centralblatt f. m. Wissensch.,' 1872.

bral arteries, and from these, and the posterior cerebral, the blood-supply of each hemisphere is derived. From the circle of Willis, and the commencement of the three cerebral arteries, small branches arise which supply the central ganglia of the hemisphere and the adjacent white substance, while the three arteries ramify over the surface of the brain, and supply the grey cortex and the greater part of the white substance of the hemisphere. Between these central and cortical systems there are no anastomoses, nor do the central branches communicate with each other. The anastomoses between the several cortical branches seem to vary in different persons. In many the communications are too slight to permit of the establishment of a collateral circulation. In others they appear to be sufficient to maintain nutrition. Moreover, similar differences seem to exist among the cortical branches in the same individual, and even among those of the same vessel; hence the effect of obstruction of these vessels varies much.*

The *central arteries* may be divided into **six** groups, **two** medial and four lateral, two on each side.

The *anterior medial group* consists of a few twigs, inconstant, which are given off from the anterior cerebrals and anterior communicating artery. They supply the anterior extremity of each caudate nucleus. The *posterior medial group* consists of twigs given off by the posterior cerebral arteries near their origin from the basilar; these twigs pass into the posterior perforated spot, and supply the inner parts of the optic thalami and the walls of the third ventricle. The supply by these groups is insignificant as regards extent, but important in regard to position, on account of its proximity to the ventricles, into which blood, escaping from one of these vessels, may readily pass.

The lateral groups supply the chief part of the central ganglia. The *antero-lateral* group arises from the middle cerebral near its origin (in the first inch of its course), and consists of a number of small arteries which, coming off at a right angle, pass into the "anterior perforated space," and supply the caudate nucleus (except its head), the lenticular nucleus, the internal capsule, and part of the optic thalamus. These arteries are of great importance, and may be divided into two sets (Fig. 44): an *internal* set, the vessels of which pass directly through the internal segment of the lenticular nucleus to the internal capsule; and an *external*, consisting of vessels that course upwards for a short distance outside the lenticular nucleus, and then pass into its third segment, and through this to the internal capsule. The vessels extend through the internal capsule, the anterior to the caudate nucleus (except its head), the posterior to the optic thalamus (except its inner and hinder part). Hence the anterior have been called the *lenticulo-striate*, and the posterior the *lenticulo-optic* arteries. These vessels are prone to rupture, and one of the former group, which runs at the outer side of the lenticular nucleus (Fig. 44), from its size and tendency to give way, has been called by Charcot "the artery of cerebral hæmorrhage." The vessels burst chiefly outside the lenticular nucleus, and the hæmorrhage separates the nucleus from the external capsule.

The *postero-lateral group* arises from the posterior cerebral, after it has passed round the crus, and supplies the hinder part of the optic thalamus. Hæmorrhage from the rupture of these vessels is apt to invade the crus on the one side and the hinder part of the internal capsule on the other. The posterior cerebral gives branches also to the crus and to the corpora quadrigemina.

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* The conclusions of Heubner and Duret differ regarding the anastomoses of the cortical vessels; the latter could not find the anastomoses described by the former. The facts of pathology are strongly in favour of the view that anastomoses between the cortical branches occur often, but not invariably. An injection often passes beyond the limit of the distribution of the artery injected.

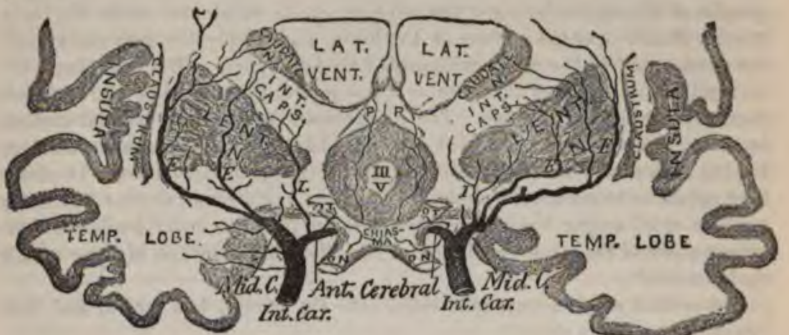
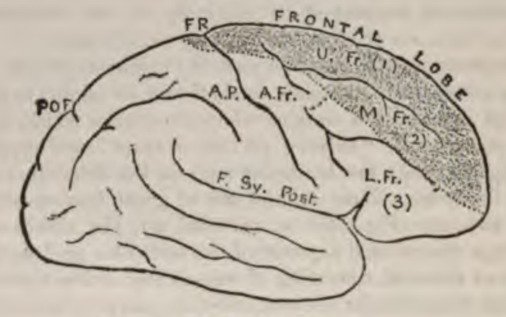


FIG. 44.—Diagram of the blood-supply to the central ganglia by the lenticulo-striate arteries, external (*E*) and internal (*I*). III V, third ventricle; P P, pillars of the fornix; *Mid. C.*, middle cerebral artery. (After Duret.)

Of the supply to the *cortex* from the three vessels, that from the middle cerebral is the most extensive and the most important, embracing as it does

FIG. 45.



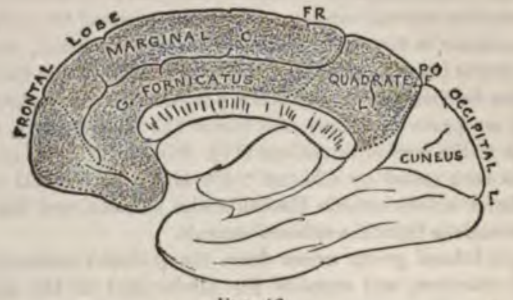


FIG. 46.

FIGS. 45 and 46.—Area of the cortex on the outer and medial aspects, supplied by the anterior cerebral artery. The supply of the several branches is indicated by the dotted lines.

the central (motor) convolutions. The general plan of distribution of each artery is the same. Each divides into certain branches, and these again divide and ramify; from the branches and the ultimate ramifications (in the pia mater) twigs are given off to the cerebral substance. These are of two kinds, long and short. The short are *cortical*, supplying only the grey cortex, in which they form a capillary network, most abundant in the middle layers which contain the large nerve-cells. The long are *medullary*, and, passing through the cortex, penetrate the white substance to various depths. The distribution of the longest reaches the neighbourhood of that of the central vessels, but the two do not blend, and at the confines of the two areas small cavities often form in old age. The cortical, moreover, do not communicate with each other, and although there is a scanty communication between the areas supplied by the larger trunks, it is often insufficient to establish a collateral circulation if one is obstructed. Hence the obliteration of branches usually (though not invariably) causes softening limited to the area supplied. This is most extensive on the surface, and lessens in extent towards the centre of the brain.

The *anterior cerebral* curves round the corpus callosum and supplies part of the orbital lobule, the inner surface as far as the quadrate lobule, and the frontal lobe, with the exception of the inferior and ascending frontal convolutions. Its chief branches are three, and supply the following parts:—(1) the supra-orbital lobule (as far as the orbital sulcus) and the inner aspect of the anterior extremity of the hemisphere; (2) the anterior half of the inner surface in front of the quadrate lobule, also the first and second frontal, and the highest part of the ascending frontal convolution; (3) the quadrate lobule (precuneus).

The *middle cerebral* divides, opposite the island of Reil, into four branches, marked I, II, III, IV, in Fig. 47. These lie in the sulci of the insula, and then, passing on to the surface of the hemisphere, have the following distribution (Figs. 47 and 48):—(1) to the third (inferior) frontal convolution; (2) to the lower two thirds of the ascending frontal and the root of the middle frontal (3) to the whole ascending parietal, superior parietal lobe, and adjacent part of the inferior parietal lobule, often also to a small part of the ascending frontal; (4) to the convolutions about the posterior limb of the fissure of Sylvius, viz. part of the inferior parietal lobule (supra-marginal and angular gyrus), posterior extremity of the superior parietal lobule, and the hinder part of the first two temporal convolutions. From this, near its origin, one or two large branches arise (V) which supply the anterior part of the first and the greater part of the

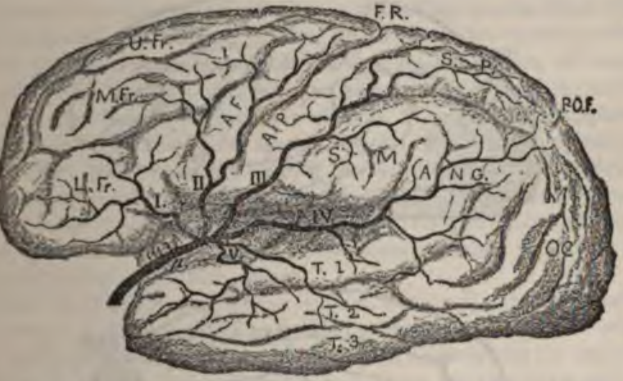


FIG. 47.—Course of the branches of the middle cerebral artery.

second temporal convolutions. There are individual variations in the precise area supplied by the branches, and also in the posterior limit of the distribution

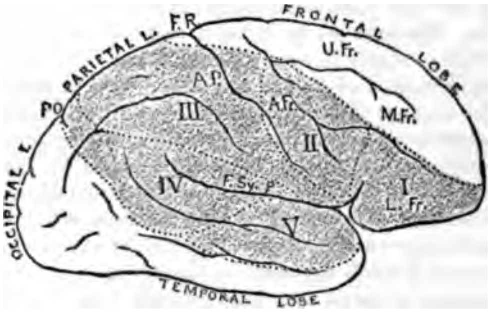
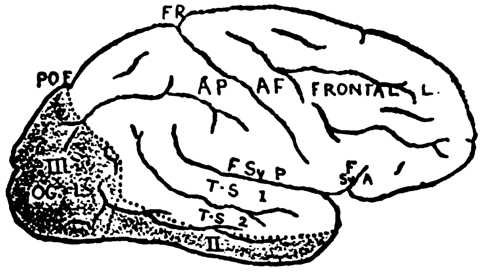


FIG. 48.—Area of distribution of the branches of the middle cerebral artery.

of the middle cerebral. This is of considerable importance, because the posterior part of the angular gyrus often escapes the softening caused by obstruction of the middle cerebral.

The *posterior cerebral* supplies the greater part of the inferior aspect of the brain, and also the occipital lobe, inner and outer surfaces, by three branches.

FIG. 49.



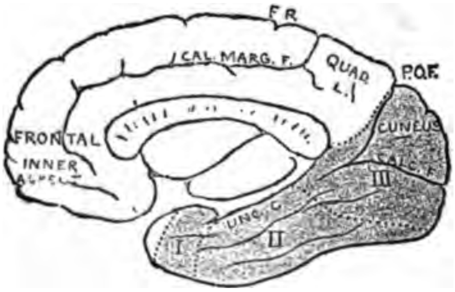


FIG. 50.

FIGS. 49 and 50.—Area of the cortex supplied by the branches I, II, III, of the posterior cerebral artery.

The vessel divides into these branches after winding over the crus, and giving off its central branches and twigs to the tegmentum of the crus, and to the corpora quadrigemina. Its branches supply (1) the lower part of the uncinate convolution; (2) the inferior part of the temporal lobe; (3) the cuneus, lingual convolution (*i. e.* the posterior part of the uncinate gyrus beneath the calcarine fissure), and also the outer surface of the occipital lobe.

Thus the middle cerebral supplies the chief part of the motor convolutions and the motor central structures, as well as the cortical auditory centre, and the greater part of the angular region. The posterior cerebral supplies the optic ganglia (optic thalamus and corpora quadrigemina and geniculata), and in the cortex the visual region in the occipital lobe.

The blood-supply to the pons, medulla oblongata, and cerebellum is derived from the vertebrals and basilar. The arrangement of the supply to the medulla and pons is very important on account of the centres which are here so closely grouped together.

It will be remembered that each vertebral, before it joins its fellow to form the basilar, gives off an inferior cerebellar artery and an anterior spinal artery. From all these vessels small branches enter the pons and medulla. The cerebellum is nourished, not only by the inferior cerebellar from the vertebral, and superior cerebellar from the anterior extremity of the basilar, but also by a large branch, the middle cerebellar artery, which comes off from the basilar about the middle of its course, and passes outwards over the pons and middle cerebellar peduncle.

The arterioles for the pons and medulla consist of two sets. (1) A series of *medial* vessels enter near the middle line in front, and pass directly back, close to the raphe, to the nerve-nuclei, which receive from these arteries the chief part of their blood-supply; (2) *lateral or radicular* vessels pass to the nerve-roots, and on each root an artery divides into a descending branch, which extends along the nerve, and an ascending branch, which enters the medulla with the nerve-fibres, and accompanies them as far as their nucleus, giving off branches as it proceeds. But the blood-supply to the nucleus derived from the radicular branch is far less than that from the median. The vertebral supplies the radicular branches to all the bulbar nerves, except a branch to the spinal accessory nerves which comes from the inferior cerebellar, and to the hypoglossal, from the anterior spinal artery. The branches to the facial and auditory nerves may come from the vertebral or basilar, or both. All the other radicular branches are derived from the basilar, but the middle cerebellar artery sometimes gives branches to the auditory and fifth nerves.

The median branches form a continuous series, but are divided by Duret into four sets, termed, from their position, *bulbar*, *subpontine* (at the lower edge of the pons), *medio-pontine* (along the pons), and *supra-pontine* (at the upper edge of the pons). The lower bulbar branches come from the anterior spinal, and supply the nuclei below the calamus scriptorius—the hypoglossal and accessory. The subpontine, from the basilar, supply the pneumogastric, glosso-pharyngeal, and auditory nuclei, while the medio-pontine and supra-pontine branches supply the nuclei in the upper half of the floor of the fourth ventricle, as well as the sixth. The anastomoses of these vessels are unimportant.

It is necessary to remember that the larger trunks about the medulla present frequent variations, which entail corresponding variations in the origin of the nutrient arteries. The left vertebral is usually larger than the right, and the latter may be very small, so that the basilar is practically a continuation of the left vertebral only. There is usually only one anterior spinal artery, arising from both vertebrals, or arising from the left only. If there are two, they usually communicate, but the left occupies the medial furrow and furnishes the median nutrient arteries. These variations explain why obstruction of one vertebral sometimes affects the nuclei on one side, sometimes on both, and sometimes on neither.

Branches to the olivary body and anterior pyramids come from the vertebral or anterior spinal; those to the restiform body arise from the inferior cerebellar, and this also gives off the posterior spinal arteries, from which branches pass to the posterior pyramids and to the upper part of the central canal, chiefly at the level of the decussation of the pyramids.

The chief *cerebellar arteries* are (1) the *posterior* from the vertebral (sometimes from the basilar), which winds round the medulla between the hypoglossal and pneumogastric nerves, and, after giving origin to the posterior spinal and branches to the choroid plexus of the fourth ventricle, supplies the inferior vermiform process and the posterior region of the hemisphere; (2) the *median* (Duret), which leaves the basilar at the middle of the pons and passes outwards over the middle peduncle of the cerebellum to ramify on the under surface; (3) the *superior cerebellar*, arising from the basilar close to its bifurcation, curves round the pons, and supplies the anterior and upper parts of the hemisphere. These cerebellar arteries communicate with each other, and the superior is often connected by one or more twigs with the posterior cerebral.

VENOUS CIRCULATION.—The veins of the convexity of the hemisphere, and of its inner surface, ascend and open in a forward direction into the superior longitudinal sinus. One of the veins of the convexity courses at first along the fissure of Sylvius, and afterwards ascends across the hemisphere: it has been termed the "Sylvian vein" by Duret.' The course of the surface veins is important because it helps to explain the frequency with which clots form within them. Elsewhere the blood from ascending arteries passes into descending veins, so that the feeble pressure through the capillaries is supplemented by the influence of gravitation. Elsewhere ascending veins convey blood that has been brought by descending arteries, and the venous pressure is aided by the "hydrostatic pressure," which tends to make the blood rise in the veins to the level of the heart. But on the brain, blood from ascending arteries passes into ascending veins. The openings of these veins into the longitudinal sinus being directed forwards, the entering blood is opposed in direction to the current in the sinus, and the effect must be to retard the flow in both veins and sinus. Moreover, in the erect posture, the anterior part of the longitudinal sinus has also an ascending course, while the trabeculæ that occupy the lumen of the sinus must offer some hindrance to the movement of the blood. These circumstances help us to understand the readiness with which clots form in the cortical veins and longitudinal sinus, when other circumstances favour the coagulation of the blood. Indeed, the marvel is that thrombosis is not more common than it is.

The veins of the inferior surface of the brain enter—the anterior the cavernous sinus, the middle the petrosal sinuses, the posterior the lateral sinus. The veins of Galen, from the ventricles, receive not only the blood from the ventricular walls and choroid plexuses, but also from the corpus callosum. The veins of the upper surface of the cerebellum open into the veins of Galen or the straight sinus in which the veins of Galen end. Those of the inferior surface open—the posterior into the lateral sinus, and the anterior into the occipital sinuses. The individual veins have not, as a rule, free communications with those adjacent, although differences exist in this respect. But the system of the convexity (superior longitudinal sinus) has three important communications. A large vein always passes from the Sylvian vein to the superior petrosal sinus

(Trolard), and another, at the base, connects the commencement of the Sylvian vein with the basilar vein, and thus with the straight sinus. The vein of the corpus callosum (going to the veins of Galen) often communicates freely with the veins on the inner surface of the hemisphere, which go to the superior longitudinal sinus. The communication between the various sinuses, with the exception of the superior longitudinal, are free. Thus the two cavernous sinuses are connected by the circular sinus around the pituitary body, and each cavernous sinus has a double communication (by the two petrosal sinuses) with the lateral sinus and jugular vein, while the two inferior petrosal sinuses are connected by the "transverse sinus" across the basilar process of the occipital bone. The cavernous sinus receives the ophthalmic vein, the superior petrosal sinus receives veins from the internal ear, and into the lateral sinus some mastoid veins open. The occipital sinus receives the spinal veins, and many sinuses receive veins from the diploë of the skull.

There exist certain communications between the cerebral sinuses and veins outside the skull. The following are the chief. Veins from the nose enter the anterior extremity of the superior longitudinal sinus. The ophthalmic vein (cavernous sinus) communicates freely with the facial vein. By the veins of the mastoid cells the occipital vein communicates with the lateral sinus. Moreover, small "emissary" veins pass through minute foramina in the cranium and connect certain sinuses with external veins. The most important of these are between the superior longitudinal sinus and the veins of the scalp, between the cavernous sinus and the pterygoid plexus of the internal maxillary vein, between the inferior petrosal sinus and the deep veins of the neck. A further communication, very variable in degree, is established by the veins of the diploë. Lastly, the spinal system, which has free external communication, is connected by six veins with the sinus system of the brain. The cerebral veins and sinuses have no valves. These facts are important in regard to the external signs of thrombosis in the sinuses within the skull.

SECONDARY DEGENERATION IN THE BRAIN.

We have already seen that some tracts of fibres in the brain undergo degeneration after a lesion in their course or in the grey matter from which they spring. In the latter case the whole length of the fibre degenerates, in the former only that portion which lies on the distal side of the lesion, *i. e.* on the side farthest from the nerve-cell which gives origin to the fibre. This secondary degeneration proceeds until the fibres are again interrupted by grey matter, and there it ceases. The nature of secondary degeneration was considered in vol. i, where we saw that the axis-cylinder of each nerve-fibre must be regarded as the prolonged process of a nerve-cell, depending on that nerve-cell for its nutritional integrity, and that the nerve-fibre therefore undergoes degeneration when the cell is destroyed or the fibre is cut off from the cell. As a general rule the direction in which a fibre degenerates is that in which it conducts; that is, the cell on which the nutrition of the fibre depends, the cell of which it is the prolonged process, is the cell from which the nerve impulses originate that traverse the fibre, or, in other words, it is this cell only which excites the functional activity of the fibre. But this rule of the identity of the direction of conduction and degeneration does not seem to be true of all nerve-fibres. As far as is yet known, it is true of all motor fibres, but there is a conspicuous exception to it in the case of the fibres of the peripheral sensory nerves. These conduct centripetally, but degenerate centrifugally from the ganglion on the posterior root. Above the ganglion they conform to the general law. In the central nervous system we may take the direction of degeneration as presumptive but not as absolute proof of the direction of conduction. It is doubtful whether all nerve-fibres undergo secondary degeneration. The fibres of the corpus callosum apparently do not. Possibly some fibres pass between two nerve-cells, and are so related to both (connecting, for instance, undivided processes) that either cell is able to maintain the nutrition of the fibre.

The most important and extensive secondary degeneration is that of the fibres of the pyramidal tracts, proceeding from the central convolutions. The course of these fibres has been already fully described (p. 28). The lesion causing their degeneration may be in the cortex, or anywhere in the course of the fibres. If it is above the crus, the degeneration appears at the under surface of the crus, in its middle two fifths (Fig. 51), and is wider above, near the optic tract, than below, near the pons, where the pyramidal fibres are overlapped by those on each side. The pons is reduced in size on the side of the degeneration, and the anterior pyramid of the medulla is small and grey. To cause extensive degeneration of these tracts, a lesion of the

FIG. 51.

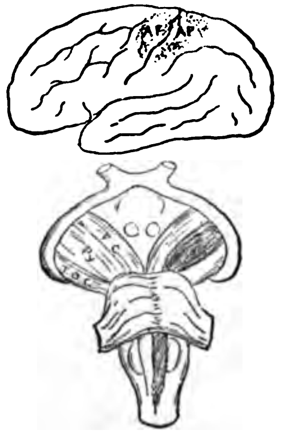


FIG. 52.

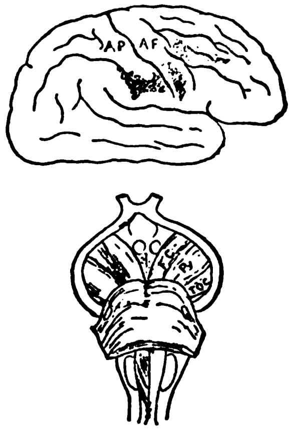


FIG. 51.—Lesion of the upper part of the central convolutions, causing descending degeneration of the pyramidal tract in the crus and the anterior pyramid of the medulla. (After Brissaud.)

FIG. 52.—Partial disease of the central convolutions, causing partial degeneration of the pyramidal fibres in the crus and medulla. More degenerated fibres come to the surface near the pons than near the tract.

cortex must be also extensive, such as that shown in Fig. 51. A slighter lesion (Fig. 52) causes partial degeneration; some fibres are involved and others escape. A lesion of the marginal convolution in the monkey is followed by scattered degeneration in the internal capsule behind its bend, but not in front of this, and in the pons and cord, where it is more compact. Lesions of the gyrus fornicatus are followed by similar degeneration in the pons and medulla, but, in the cord, this is spread out over the whole transverse sectional area of the crossed pyramidal tract of the opposite side alone, and gradually diminishes in amount from above downwards. (France, 'Phil. Tr.,' 1889.)

A lesion within the pons causes degeneration only of

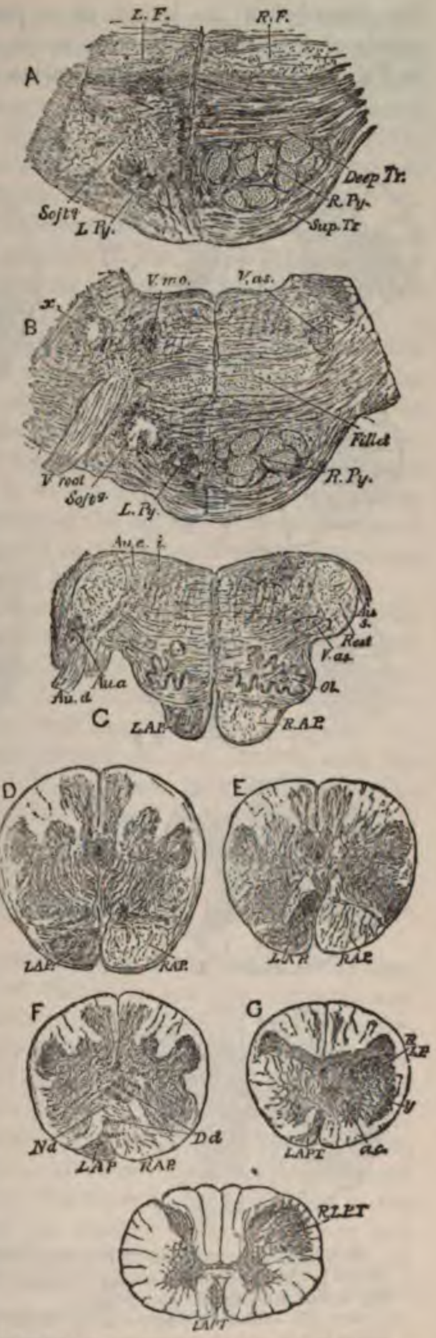


FIG. 53.—Softening in the left side of the pons in the crustal portion (A), and near the nucleus of the fifth nerve (B *x*). The other figures represent sections through various parts of the medulla and decussation of the pyramids; the last is of the spinal cord. *L Py*, *R Py*, left and right pyramidal tract; *L F*, *R F*, left and right fillet; *Deep Tr*, *Sup Tr*, deep and superficial transverse fibres of the pons; *V mo*, *V asc*, motor nucleus and ascending root of the fifth nerve; *Au d*, *Au s*, deep and superficial roots of the auditory nerve; *Au*, *e*, *i*, *a*, its external, internal, and anterior nuclei; *Rest*, restiform body; *L A P*, *R A P*, left and right anterior pyramid; *D d*, degenerated (left), *N d*, not degenerated (right) pyramidal fibres at the decussation of the pyramids; *L A P T*, left ant. pyr. tract; *R L P T*, right lateral pyr. tract; *a c*, anterior cornu; *y*, slight degeneration in the anterior part of the lateral column.

the fibres below the lesion, as in the case shown in Fig. 59. The effects of the area of softening in the crustal half of the pons, shown in Fig. 58, present a good illustration of the downward course of the

FIG. 54.

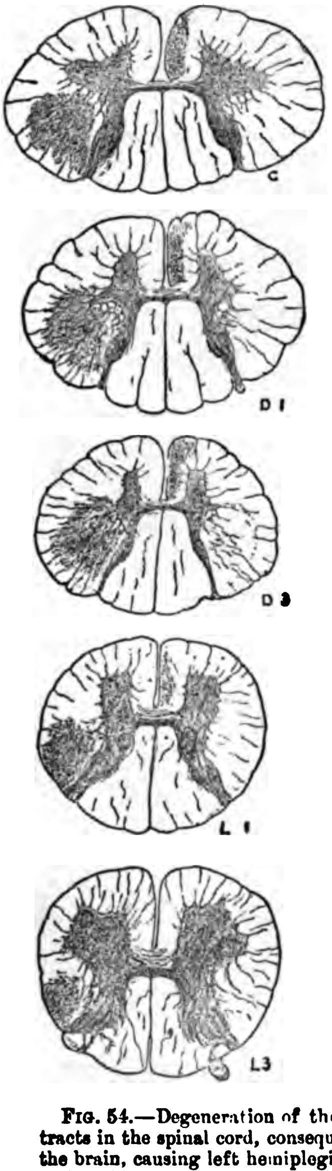
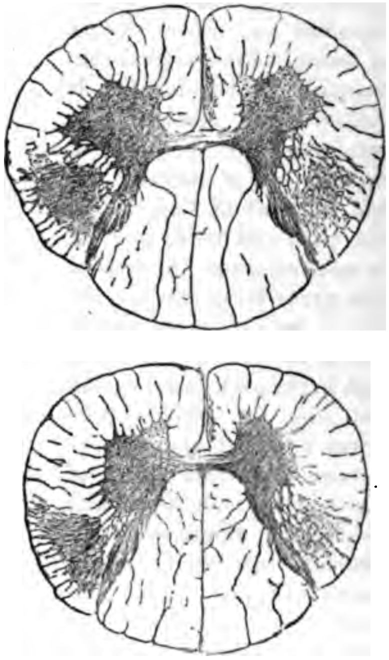


FIG. 55.



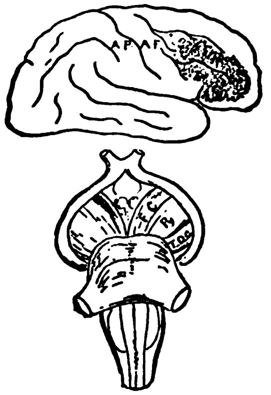


FIG. 56.

FIG. 54.—Degeneration of the right anterior and left lateral pyramidal tracts in the spinal cord, consequent on a lesion in the right hemisphere of the brain, causing left hemiplegia.

FIG. 55.—Bilateral degeneration of the pyramidal tracts secondary to a lesion in the right hemisphere. (From sections by Prof. Pitres.)

FIG. 56.—Area of disease of the right pre-frontal lobe, causing partial degeneration of the inner fibres of the crusta.

degeneration through the medulla and the decussation of the pyramids. In Fig. B the degenerated bundles of the pyramidal tract are separate, but in C and D they are united in the anterior pyramid, which is completely degenerated. The passage of the degenerated fibres through the decussation of the pyramids is shown in F and G; in the latter most of them have reached the opposite lateral column of the cord, and occupy the position of the lateral pyramidal tract, while the degeneration in the anterior column is reduced almost to the small dimensions which it presents in the lowest figure (of the cord). The descending changes in the spinal cord have been described in the first volume, and are shown in Fig. 54.

In some cases of disease of the motor path in one hemisphere there is a double degeneration in the cord, a slight degeneration in the lateral pyramidal fibres on the side of the cerebral lesion, as well as considerable degeneration in the lateral tract of the opposite side. Attention has been called to the frequency of this double degeneration by Pitres.* An example of it is shown in Fig. 55. It is, perhaps, related to the double representation of the leg in both hemispheres of the brain, which will be mentioned in the account of the symptoms of brain disease. It is not present, however, in all cases.

Another frequent secondary degeneration is that of the fibres which course from the pre-frontal lobe of the brain (in front of the central convolutions) through the anterior limb of the internal capsule, and the inner part of the crusta of the cerebral peduncle to the grey matter of the pons. In the pons their degeneration ceases, but the evidence that they are connected, through its grey matter, with the cerebellum is very strong (see p. 32). Hence they are termed the fronto-cerebellar fibres (F C, Fig. 56). Their degeneration is visible in the most internal portion of the crus, and may be produced by disease either of the frontal lobe in front of the ascending frontal convolution (see Fig. 56) or of the anterior limb of the internal capsule (Fig. 57).

Degeneration of the most external fibres of the crusta, those that pass between the grey matter of the pons and the temporal and occipital lobes, has been met with in rare cases. In an instructive case of congenital atrophy of one occipital lobe, extending as far as the middle of the posterior limb of the fissure of Sylvius, there was also atrophy of the pulvinar, the optic tract, the external geniculate body, the superficial layer of the anterior quadrigeminal body, and its anterior brachium.† Degeneration of those fibres has been caused by extensive damage to the cortex (Bechterew), or by a lesion in the vicinity of the hindmost fibres of the internal capsule, as in the case shown in Fig. 58. It is not the disease of the capsule itself which causes the degeneration, for the fibres that run in this part of the capsule proceed from the tegmentum (p. 32), and probably degenerate, as they certainly conduct, upwards. The degeneration is due to the damage to the adjacent fibres, which, as they leave the crus, do not enter the capsule, but pass close by it, to radiate to the temporal and occipital cortex.

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* See also Hadden and Sherrington, 'Brain,' 1886.

† Kreusner, 'Allg. Zeitschr. f. Psych.,' Bd. xlviii.

Fig. 57.

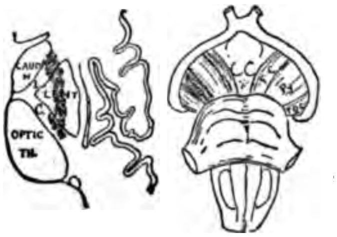

Fig. 58.

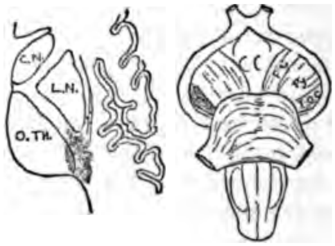

Fig. 59.

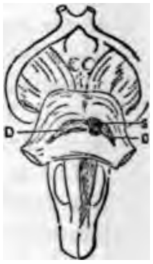

Fig. 57.—Lesion of the lenticular nucleus and of both limbs of the capsule, causing partial degeneration of the inner and pyramidal fibres of the crusta.

Fig. 58.—Disease of the posterior part of the capsule, damaging also the adjacent fibres from the outer part of the crusta, which have undergone secondary degeneration. (After Brissaud.)

Fig. 59.—A lesion (S) of the pons, causing secondary degeneration of the transverse fibres (D D) and of the anterior pyramid of the medulla.

The transverse fibres of the pons are said to degenerate when interrupted by a lesion in their course. Of adjacent fibres, some seem to degenerate in one direction, and other fibres in the opposite direction, so that the degeneration extends on each side of the lesion, as in Fig. 59. In the case shown in Fig. 53 such degeneration could not be distinctly traced.

The above are the chief secondary degenerations that are of medical significance. Some other degenerations have been met with, such, for instance, as that of the fillet, but they are not yet of sufficient definiteness to warrant their detailed description.

THE SYMPTOMS OF BRAIN DISEASE.

The symptoms produced by diseases of the brain are, for the most part, the same in character, whatever be the morbid process that causes them. They depend on the seat of the disease, and on the nature of the change that the nerve-elements undergo, rather than on the pathological character of the primary lesion. In most diseases that we term "organic" the primary morbid process is outside the nerve-elements themselves; the changes these elements undergo are secondary, and are few in kind, while the diseases by which they are produced are numerous. In the maladies that we term "functional" (which are better conceived as "nutritional," see vol. i, p. 2) there is no lesion outside the nerve-elements; the change which these undergo, and on which the symptoms immediately depend, is primary. This is probably the case also in the degenerations of the brain, although the changes in the nerve-elements, and those in the interstitial tissue, often seem to occur almost at the same time.

The symptoms of disease show that there is disorder of function, either "objective" and recognisable by another person, or "subjective" and perceived only by the sufferer himself. The disturbance of function is of two kinds: (1) a diminution of action, which may present every degree down to abolition; and (2) an increase of action. These two effects are often combined. Disease may lessen the possible amount of functional action, and yet render that which remains too readily excited. This is the "irritable weakness" of old writers. The increase of action caused by disease is commonly called "irritation." But irritation sometimes lessens function by the process that physiologists term "inhibition." Thus loss of function may be due to structural damage or to irritation; from the latter the loss is transient, from the former it is permanent. Lastly, function may be gravely deranged and even arrested by a change in the blood, especially by the presence in it of new elements, constituting what is termed a "toxæmic" state. The facts at present available suggest that these agents may be either organisms or the products of their growth—perhaps merely the chemical products.

Structural damage to the nerve-elements is produced in various ways by organic disease of the brain. The following are the most important mechanisms:—(1) Mechanical interruption or destruction, as when nerve-fibres are torn across, or nerve-cells are separated by an extravasation of blood. This involves, of necessity, entire abolition of function. (2) Mechanical compression, which may be sudden or slow in its production, and varies in its influence on function according to its rapidity. Very slight compression, if suddenly or rapidly induced, arrests function; on the other hand, a considerable amount of pressure produces little or no effect if it be very slowly induced. We do not know whether pressure acts on function by a mechanical influence on the nerve-elements, or by an arrest of the blood-supply due to compression of the capillaries. The difference between the effect of slow and sudden pressure suggests that its influence on the nerve-elements is direct, since the effect on the capillaries must be the same in each case. Pressure does more than arrest function; it causes structural disintegration of nerve-elements, and this also is greater when the pressure is rapidly produced. (3) Arrest of the blood-supply causes arrest of fuction, and diminution of the blood-supply causes impairment of function. Here also we may trace a relation to rapidity of production, although slighter than in the case of pressure, and perhaps due to the fact that slow obstruction gives time for the establishment of a collateral circulation. The immediate arrest of function that follows a sudden arrest of blood-supply is very remarkable. If the heart fails, for instance, consciousness is instantly lost. It is probable that the influence is in part mechanical, due to the sudden diminution in the pressure to which the nerve-elements are normally exposed. It is probable, therefore, that a decrease, as well as an increase, of pressure interferes with function. (4) Inflammation—which, in the brain as in other tissues, involves the interstitial tissue and the proper elements of the part, as well as the blood-vessels. (5) Molecular disintegration or wasting of the nerve-elements. This constitutes the primary and chief element in many degenerative processes, in which it is associated with changes in the interstitial tissue. It occurs also, in rapid form, in consequence of many of the other morbid processes above described.

Some symptoms, such as local paralysis, are due to and indicate interference with the function of a definite part of the brain. They are often termed *focal* symptoms. Other symptoms, such as loss of consciousness or delirium, indicate a wide-spread interference with function, and are called *diffuse*. The distinction, although useful, must not be conceived as absolute. Some symptoms may be at one time diffuse, at another focal. The same terms are also applied to the morbid processes, but the two applications do not altogether correspond. Diffuse processes may cause focal symptoms, as when hemiplegia results from an inflammation extending over the whole of one hemisphere. On the other hand, a focal lesion may cause a diffuse symptom, as when a small hæmorrhage produces loss of consciousness.

It is necessary, in the first place, to describe the characters of the several symptoms of organic disease of the brain. Some of these symptoms consist of disturbance of the functions of the cranial nerves, and it will be convenient to describe them in connection with the diseases of those nerves, before we consider the general rules of diagnosis and the special diseases of the brain.

Motor Symptoms.—Motor Paralysis—Hemiplegia.—Loss of voluntary power over the muscles is one of the most frequent and important effects of brain disease. A distinction is often drawn between complete and partial loss of power, and the former is termed "paralysis," the latter "paresis." The two differ only in degree, and the term "paralysis" is also applied to all grades of deficiency. The loss is due to interference with the motor centres or the motor path, which, as we have seen, passes from the central convolutions down through the centrum ovale, the internal capsule, the crusta of the cerebral peduncle, and the corresponding region of the pons, to the anterior pyramid of the medulla. The disease that causes paralysis may be situated in any one of these regions. Disease elsewhere does not cause loss of motor power unless it exerts pressure on the motor tracts. The paths from the two sides are near together in the pons, and still nearer in the medulla; hence a lesion in either of these situations may paralyse both sides of the body. Even in the pons, however, a single lesion often affects one side only, while in the crura the paths diverge, and both suffer only when they are damaged by a large tumour. In the cerebral hemispheres a single lesion always affects only one motor path. The unilateral paralysis that results from disease of the tract on one side is termed "hemiplegia." The paths decussate in the medulla, and therefore the limbs are paralysed on the side opposite to the lesion in the brain. In the pons the motor paths, before crossing the middle line to the hypoglossal and facial nuclei, are associated with the path from the limbs, and hence a lesion anywhere above the middle of the pons usually causes also paralysis of the face and tongue, which is on the same side as the palsy of the limbs. The motor fibres diverge in the internal capsule, and still more as they pass through the white substance to the cortex, where, as we have seen, are centres, to some extent separate, for the leg, arm, face, and tongue. Hence lesions of the cortex, or of the white substance just beneath it, may paralyse one of these parts without the others; but such partial palsy seldom results from a lesion in the internal capsule, because, to have so limited an action, a lesion there must be minute.

To the rule that the paralysis is on the side opposite to the cerebral lesion, apparent exceptions have been met with, in which hemiplegia was on the same side as the lesion. Morgagni suggested that in these cases the ordinary decussation of the motor tracts does not occur; and the discovery by Flechsig that there are great variations in the decussation of the pyramids in the medulla has been regarded as confirming the hypothesis of Morgagni. But there is strong reason to believe that when the paths do not decussate in the medulla they cross lower down the spinal cord, and if so this explanation falls to the ground. Much more frequent than hemiplegia on the same side as the lesion is hemiplegia without any discoverable lesion to account for it. The nature of these cases is mysterious, but most pathologists have met with instances. They are probably due to a morbid blood-state, as already explained.

In a case of severe "complete" hemiplegia the arm and leg are powerless; the face is paralysed, chiefly in the lower part, and moves almost as well in the upper part as on the unparalysed side; the tongue, when protruded, deviates towards the paralysed side, being pushed over by the genio-hyo-glossus of the sound side. But the muscles of mastication contract equally in ordinary action, and the two sides of the thorax move equally in ordinary respiration. Some of the trunk muscles may be weakened; the patient cannot sit up. If he takes a deep breath, and brings into action the extraordinary muscles of respiration, the half of the thorax on the paralysed side often expands less than the other. Thus some muscles are completely paralysed, others are merely weakened, others are apparently unaffected. This difference in the condition of the muscles becomes greater after the lapse of a few months. Even when the motor path in one hemisphere is completely interrupted by the disease, and the interruption is permanent, the muscles that were merely weakened improve in strength, and in some others, that were at first totally paralysed, a certain amount of voluntary power returns. Some movement of the leg is regained, especially at the hip- and knee-joints; in the arm the shoulder-joint can be moved a little, and often also the elbow, although the hand may remain motionless. Such recovery, the lesion persisting, occurs to some extent at all ages, but much more readily in the young than in the old. In children the recovery of the leg is so great that in time there is ability to walk long distances, in spite of the destruction of the motor path. In adults, the palsy of the hand may remain almost absolute; often, however, slight power of flexing the fingers returns, and in children some recovery of movement in the hand is invariable, and it may ultimately be considerable, although disordered.*

This difference in the initial palsy, and the return of power in certain muscles, manifestly suggest a complex relation of the muscles to the centres of each hemisphere, and this relation is often stated by an hypothesis called "Broadbent's;" but, as stated by Broadbent, it was in a form somewhat different from that which seems best to meet the exigencies of present knowledge. Its form has indeed changed more than once, but the credit belongs to him of having first definitely stated it. Some muscles of the body, as the intercostals and masseters, are used only with their fellows of the opposite side; others are often used with their fellows, but often also alone, as the zygomatici, the trapezii, and many leg muscles; others are chiefly used alone, as the muscles of the hand. Movements are represented exclusively in the opposite hemisphere in proportion as they are unilateral, in both hemispheres in proportion as they are bilateral in execution. In other words, either hemisphere can excite the bilateral movements, but only the opposite hemisphere can excite the unilateral movements. The muscles that are sometimes used with their fellows of the opposite side and sometimes alone, which recover partially with a persistent lesion, are connected with both hemispheres, but are habitually excited from the opposite hemisphere; they recover some power because the hemisphere on the same side gains the influence over them for which structural arrangements previously existed, but had not been used.

We do not yet know the nature of the connection between the hemisphere and the muscles on the same side. Broadbent thought it was by a connection between the lower (spinal and bulbar) centres on the two sides, which enabled the bilateral muscles to be excited together from one hemisphere. It is highly probable that such a connection does exist, and it may be actually effective in the case of the bilateral use of muscles; but such a connection cannot subserve the unilateral use of such muscles, as, for instance, the isolated movement of the leg. This implies a separate path to the lower centre; a mere connection of the lower centres would not suffice. We do not know whether this separate path is by pyramidal fibres that do not decussate (as by the anterior pyramidal tract, or by the passage of some fibres into the lateral column of the same side), or whether it is by a recrossing in the cord. We must, however, associate this relation with the fact that degeneration may be found in both lateral columns when the disease is on one side of the brain (see p. 70); but there is a difference of opinion as to the way in which this degeneration arises, whether the fibres that degenerate come down from the medulla, as is suggested by Pitres' observations on man, or whether they are due to a recrossing in the cord, as Sherrington concludes from his experiments on animals.*

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* Thus, in one case of severe hemiplegia in early life, the patient became able to walk long distances, and had some power of (disordered) movement in the hand. After death in adult life the opposite internal capsule was found destroyed. A large cavity occupied the whole of the central ganglia.

But the retention of some movements and the return of others suggests another correlated fact. If bilateral movements are excited from both hemispheres, disease of one hemisphere should lessen the power on both sides. In proportion as muscles escape or recover on the side opposite to the lesion they should be weakened on the side of the lesion. As a matter of fact this is so. Most of the bilateral muscles are, however, so placed that their strength cannot be measured, but many muscles of partly bilateral use can be tested, and these are always found to be weakened on the side of the lesion. The weakening of this side was pointed out long ago by Brown-Séquard, and has been also investigated by Pitres† and Friedländer.‡ It is greater when the lesion is in the left hemisphere than when the right is diseased. In the leg the loss is always considerable, and is indeed conspicuous, even on a cursory examination. It exists also in the trunk muscles, where it is less readily detected, and it exists also in the arm in a degree for which theory scarcely prepares us. The power of the hand is often reduced to not much more than one half of the normal. Such a loss is not adequately explained by the hypothesis above stated, and seems to show that this hypothesis, however true and important, does not express the whole truth, part of which may be lost sight of, obscured by developmental processes in which it arose. This is especially true of the complementary weakening that should correspond to the preservation of strength, and which can scarcely be otherwise explained.

Some other features of hemiplegia remain to be described. Certain movements towards one side are produced by the action of muscles situated on both sides. Such, for instance, is the lateral movement of the eyes, by the external rectus of one side and the internal rectus of the other. Such also is the rotation of the head; the face is turned towards one side by the opposite sterno-mastoid, acting with other less important muscles of the side towards which the head is turned. In hemiplegia these movements towards the paralysed side may be impaired. The head and eyes cannot then be turned towards the affected side; and the unopposed antagonistic muscles may even turn the head and eyes towards the unparalysed side. This is termed "conjugate deviation of the eyes." The symptom usually passes away in the course of a few days, but occasionally continues for weeks, and is perhaps occasionally represented by nystagmus on movement in the direction concerned. From this we learn two facts. First, that movements, rather than muscles, are represented in the cerebral hemispheres, and are lost in disease. Secondly, that these lateral movements by muscles of both sides are represented in both hemispheres, but that in a normal state they are chiefly effected by the opposite hemisphere. When this is diseased they are impaired, until the hemisphere on the same side has acquired functional power over them through mechanisms before existing but unused.

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* 'Journ of Phys.,' vi, Nos. 4 and 5.

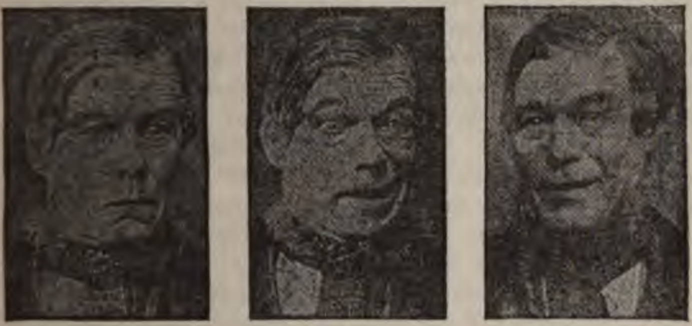
† 'Arch. de Neurologie,' 1882.

‡ 'Neur. Centralbl.,' 1883, p. 241.

A sudden cerebral lesion, such as causes hemiplegia, is often attended by loss of consciousness. We are then deprived of the direct evidence of loss of voluntary power, and a slight degree of weakness cannot be detected. But the existence of considerable paralysis can generally be ascertained. There may even be greater flaccidity of the paralysed limbs, which, if raised, fall like a weight influenced only by gravitation, while the opposite limbs, although they also fall, do so less suddenly. We are also helped in this condition by the state of reflex action, which is often though not always diminished or abolished. This is best seen in the abdominal, cremasteric, and plantar reflexes, when these are compared with the corresponding actions on the opposite side. Moreover, during the period of unconsciousness, the conjugate deviation of the head and eyes is often distinct; if the head be placed in the mid-position, the deviation slowly returns. A slight degree of ptosis may also sometimes be noticed on the paralysed side.

If the patient is conscious, the power of voluntary movement can be directly tested, and if the paralysis is not absolute the several movements of each joint should be separately examined, and the force with which passive movement can be resisted should be ascertained. The patient is almost always able to close the eye, but often a strong contraction of the orbicularis is less on the paralysed side than on the other. When there is no difference in the strength of contraction of the orbiculares palpebrarum, the patient, if previously able to shut the eye on the hemiplegic side without the other, is usually no longer able to do so. In the early stage the patient may be unable even to close the eye on the hemiplegic side. Sometimes there is even slight weakness of the frontalis, and the forehead may be smoother on the paralysed side than on the other. In the lower part of the face there may be a marked difference in the degree of affection of the voluntary, emotional, and associated movements (Figs. 60—62). The former is tested by making the patient raise the upper lip, and move the mouth from side to side; emotional movement is seen in a smile, and the associated movement is obtained by causing the patient to grasp strongly with the sound hand. This difference is occasionally of considerable diagnostic importance. In some cases, in the early stage, the eye cannot be closed, and there may be a doubt whether the paralysis is due to disease of the nerve or of the motor tract in the hemisphere. In the former case all emotional movement is lost, as well as that which is voluntary. In the latter the smile may be equal on the two sides although voluntary movement is lost on one. The movement of the tongue within the mouth should be observed, as well as its deviation on protrusion. The base of the tongue is usually higher on the paralysed side than on the other.

FIG. 60. **FIG. 61.** **FIG. 62.**



FIGS. 60—62.—Different affection of voluntary and emotional movements of the face in a case of right hemiplegia. The appearance of the face at rest is shown in Fig. 60; an attempt to raise the upper lip on both sides in Fig. 61; a smile in Fig. 62. The last was much more nearly equal on the two sides than the figure suggests. (From photographs.)

If hemiplegia is complete in extent but imperfect in degree, the condition is similar to that of perfect hemiplegia which is in process of recovery. The loss of power is greater in the arm than in the leg, and greater towards the extremity of each limb than in the part nearest the trunk. The muscles moving the hand are weaker than those of the shoulder, and in the leg the most affected movements are those of the foot, especially flexion of the ankle. Hence, in walking, the foot is swung round (or, less commonly, the knee is unduly flexed) in order to prevent the contact of the toes with the ground when the foot is brought forwards. There is often more power of using the leg with the other, as in walking or standing, than there is in the separate movements of the limb.

To the rule that the leg recovers before the arm, and the proximal parts of the limbs before the distal, apparent exceptions are sometimes met with. The arm may recover earlier and more than the leg, and sometimes the distal recover before the proximal parts of the limbs. We should remember that, in comparing the two modes of recovery, we are (or may be) comparing things in their nature unlike —the return of power due to compensation by the other hemisphere, with that due to the recovery of the damaged hemisphere.

Varieties of hemiplegia depend, for the most part, on differences in the seat of the lesion. These differences may be spoken of as transverse and vertical. The transverse variations determine differences in the distribution of the palsy on the affected side, and are greater the higher the lesion is in the motor path, reaching their maximum at the cortex. The vertical differences determine chiefly the associations of the hemiplegia, although, as we have just seen, they are not without influence on its distribution, on account of the different compactness of the path, and consequent differences in the scope for transverse variations in the lesion. Where the path occupies a small transverse area, a single lesion can hardly fail to influence the whole of it.

The varieties determined by the relative amount of damage to the several parts of the motor path or centres depend on the distribution of the palsy, whether this is "complete" from a total lesion, or "incomplete" from a partial lesion. The latter occurs chiefly when the disease is in the white substance of the hemisphere or in the cortex. Such limited palsy is termed "monoplegia,"* or "dissociated hemiplegia." The arm only, the leg only, or the face and tongue may be affected. The two last named are usually involved together in consequence of the proximity of their centres and path. These varieties have been termed *brachial, crural,* and *facio-lingual monoplegia.* The face and arm may also suffer together (brachio-facial monoplegia). The paralysis is never absolute in these cases of limited range, and the condition of the limb, as regards loss of power, usually resembles that of hemiplegia which is in process of recovery. The coarse movements of the upper part of the limb are preserved, while the movements of the hand may be lost.

The second class of varieties depend on the vertical position of the lesion. If this is in the medulla the face escapes altogether, or only the lips are involved. Disease at the level of the hypoglossal may, by damaging the fibres of this nerve, cause its paralysis on the same side as the lesion, and a deviation of the tongue towards the unparalysed side. Theoretically, hemiplegia without paralysis of face or tongue may result from a lesion of one pyramid below the hypoglossal nuclei, but such cases are extremely rare. In the lower part of the pons the facial nerve is damaged on the side of the lesion, and therefore on the side opposite to the palsy of the limbs. In the upper part of the pons the fibres from the opposite facial nucleus have joined the motor tract, and the hemiplegia does not differ from that produced by disease of the internal capsule, unless the fifth nerve is involved on the side opposite to the limbs. If the lesion is in the crus, the fibres of the third nerve may be damaged on the side of the lesion, and there is, in addition to the hemiplegia, palsy of the third nerve opposite to the hemiplegia, usually complete, sometimes incomplete, and affecting especially the levator. The impairment of sensation that often accompanies hemiplegia will be described in a separate section.

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* Strictly, "monoplegia" ought to be used to designate double hemiplegia, but this is commonly called diplegia. We have thus the anomalous nomenclature (too firmly rooted to be disturbed) that two half-palsies make a double palsy, and that a one-palsy is part of a half-palsy!

Associated movements sometimes occur in the parts paralysed. An effort to grasp with the unaffected hand will sometimes cause a similar movement in the paralysed hand. More common still is a movement of the paralysed arm during the act of yawning or stretching, or even during coughing. When partial recovery of power has taken place, voluntary movements of the affected limb may cause an associated movement of the unaffected limb. The explanation of these associated movements is doubtless to be found in the connections of the subsidiary centres through which they are produced.

The diminution in the superficial reflex action already mentioned is almost invariable at first. Sometimes it passes away in the course of weeks or months; in other cases it persists. In rare cases an increase in the superficial reflex action has been met with. This difference bears no relation to the change in sensibility. We do not yet know the significance of the difference. Some physiological researches suggest that there is a cerebral centre which normally restrains the activity of the spinal reflex centres, and that this is, in its turn, controlled by a higher centre, the influence of which may be cut off by the lesion. In consequence, the reflex inhibitory centre is unrestrained, and lowers reflex action on the paralysed side. This loss of reflex action is of diagnostic importance, because it often occurs when the motor paralysis is slight.

In rare cases, considerable movements of the limbs, even of the arm, similar to the associated movements, may be produced by cutaneous stimulation. The effect may not be confined to the paralysed side. Thus, Nothnagel mentions a case in which a moderate pinch of the hemiplegic arm caused muscular contractions in the other arm; on a stronger pinch the contractions extended to the other leg, and on a still stronger pinch, to the leg also on the same (the hemiplegic) side, whilst the arm that was pinched always remained still.*

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* 'v. Ziemssen's Handbuch,' Bd. xi, 1, ii Auflage, p. 110.

Muscular Rigidity.—At some period the paralysed limbs usually present rigidity of the muscles, which stiffens the limbs in a certain posture, and opposes passive movement. The attempt to overcome the rigidity by forced extension of the muscles causes pain. Todd first drew a distinction between two forms, "early" and "late" rigidity, and to these we must add two others. (1) *Initial rigidity* comes on immediately after the onset, and often lasts for a few hours only. It is due to the irritation of the fibres by the process that interrupts their continuity, and is often absent. The (2) *early rigidity* comes on within a few days of the onset, and lasts for a few weeks. The posture which the limbs assume is that of rest. It is apparently due to the irritation of the fibres of the motor tract by the inflammation that the lesion sets up. It is usually slight in degree, but sometimes considerable, when there is an unusual amount of irritation. If the initial rigidity is prolonged it may pass into the early rigidity. The (3) *late rigidity* comes on in the course of a few weeks, and lasts for months or years; in cases of enduring paralysis it may be permanent. This rigidity occurs when there is descending degeneration in the motor (pyramidal) tracts, and seems to be influenced in its degree by the irritative nature of this degeneration (Charcot). In the upper limb the position is that of adduction of the shoulder, flexion of the elbow, flexion and pronation of the wrist, and still greater flexion of the fingers, especially of the distal phalanges, by the contraction of the long flexor (fig. 63), the interossei, which flex the metacarpo-phalangeal joints, being little affected in this form of contracture. When the wrist is passively flexed the fingers can be extended freely, but if the wrist is extended they return to the state of flexion, because extension of the wrist lengthens, and flexion shortens the course of the flexor tendons (fig. 64). Although the contracture preponderates in the flexor muscles, the extensors present also some rigidity, as passive movement readily demonstrates. Very rarely the fingers are extended at all joints (including the metacarpo-phalangeal), but in these cases there is never complete paralysis, and the rigidity also is slight. In the leg the rigidity is more nearly equal in the two groups of muscles, and produces extensor contracture, so that the leg is straight, and the foot tends to assume the position of talipes equino-varus. The face does not participate in this form of rigidity.

The late rigidity depends on an active muscular contracture. It lessens very much during sleep, and when the limb is placed in warm water. It can also be overcome by passive extension, most readily when this is gentle and long continued. The relaxation is facilitated by rubbing the muscle. Faradisation of the opponents of the most contracted muscles also lessens the rigidity. When late rigidity has continued for a considerable time, tissue changes sometimes take place in the shortened muscles, in consequence of which their passive elongation is no longer possible. Thus we must distinguish from the active late rigidity an ultimate (4) *structural contracture.*

The rigidity, early and late, evidently depends on the over-action of the spinal centres. The centres which thus overact are probably those on which normal muscular "tone" depends, and the rigidity is an excessive degree of this "tone." With it is associated an excess of the peculiar muscular irritability on which the so-called "tendon reflexes" depend, due probably to a muscle-reflex action (see vol. i, p. 15). The excess is not usually evident until a week or ten days after the onset of the hemiplegia. In consequence of this increased irritability there is, in most cases of hemiplegia with persistent weakness, an excessive knee-jerk, and a foot-clonus can generally be obtained. Sometimes a rectus-clonus can be elicited. The same condition may be observed in the arm. A tap on the front of the wrist causes a contraction in the flexors of the fingers; one of the radius produces a contraction in the biceps, one on the ulna a contraction in the triceps, and this can be still better obtained by a tap on the tendon of the triceps just above the olecranon. A clonus can sometimes be obtained in the flexors of the fingers by putting sudden tension on them. I have even obtained a similar clonus in the trapezius by sudden depression of the shoulder.

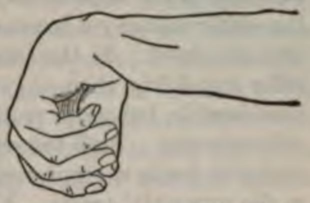

FIG. 63.—Late rigidity in hemiplegia five months after the onset.

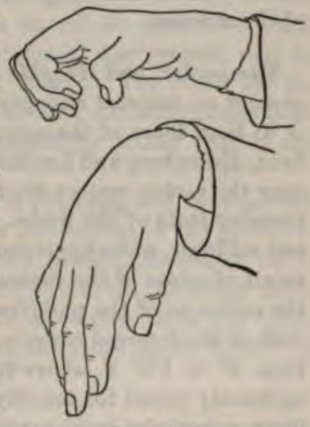

FIG. 64.—Late rigidity in hemiplegia, showing the flexion of the middle and distal phalanges when the wrist is extended, and their extension when flexion of the wrist shortens the course of the tendons.

The increase of myotatic irritability, which depends on degenerative changes in the pyramidal tracts, comes on, as has been said, about ten days after the onset of the hemiplegia. But the myotatic irritability sometimes presents variations before the degenerative increase is established, and these are probably due to the cerebral irritation, which may increase or inhibit the action of the spinal centres on which the phenomena depend.

Indeed, immediately after the onset of hemiplegia, the knee-jerk may be absent, and it may return in a few hours. When it is thus lost there is perfect relaxation. More frequent than this loss is an initial increase; soon after the onset the knee-jerk is increased, and a foot-clonus can be obtained. This early increase may or may not pass away before the degenerative excess is developed.

Muscular Irritability and Nutrition.—In cerebral disease there is rarely any considerable change in the irritability of the muscles. It may be the same as that on the unparalysed side, or it may present a slight change, increase or diminution. The change is never considerable, and is the same to both faradism and voltaism. The increase is the earlier change; a decrease is not usually present until some months after the onset. In the cases in which there is an alteration of muscular irritability, there is generally wasting of the muscles, sometimes considerable, but never reaching the degree seen in progressive muscular atrophy. Both the wasting and the alteration in irritability are probably due to the irritative character of the descending degeneration in the pyramidal tracts. Although the degeneration does not spread to the motor nerve-cells as a destructive process, it seems, when irritative in character, to influence their nutrition and the nutrition of the motor nerve-fibres proceeding from them, and, through these, that of the muscles.

Vaso-motor and Trophic Changes.—The paralysed limbs (1) may present no vascular changes; (2) they may be warmer by half a degree or so than those of the opposite side; (3) they may be colder, pale or livid. Eulenberg and Landois have ascertained that the cortex contains, near the motor centres for the limbs, other centres that influence the vascular state of the limbs. Irritation of these centres causes pallor and coldness, while hyperæmia and increased warmth are probably the result of a loss of the central influence, and may be due to disease of the cortex or of the path from it, which seems to pass in the posterior limb of the internal capsule. The elevation of temperature may be from ·2° to 1·5° F. above that of the unparalysed side. It is often uniformly raised for ten days or a fortnight, and then presents variations, sometimes an elevation, sometimes a depression. The elevation of temperature may be accompanied by redness of the skin; when the temperature falls the vessels may contract, causing pallor, or may remain distended, and the limb then has a bluish-red aspect. Occasionally there is increased sweating. There may be some subcutaneous œdema, most marked towards the extremity of the limb. This, in slight degree, is very common, and may come on at the end of one or two days and persist for many weeks. It is especially great when there is kidney disease, and may be very marked in the paralysed limb and absent elsewhere. In some cases there is a tendency to acute trophic changes. Slight pressure, continued for a few hours, may raise a blister, and even cause the skin to slough; sometimes, indeed, the vesication and sloughing seem spontaneous. Bedsores thus readily occur. Their most common seat in hemiplegia is the gluteal region, while in paraplegia it is usually over the sacrum. The skin over the trochanter, malleolus, and heel is also apt to slough, manifestly because these points are the most exposed to pressure. A marked tendency to such sloughing is of bad prognostic significance.

More chronic trophic changes are sometimes observed. Dissemiuated neuritis, with considerable thickening of the nerves at certain spots, has been met with (Leubuscher), and is said to be independent of degeneration in the pyramidal tracts (Charcot). Inflammation of the larger joints, attended with redness and swelling, has been occasionally observed during the latter part of the period of inflammatory reaction, three or four weeks after the onset. It is limited to the hemiplegic side, and is evidently analogous to the similar inflammation that is secondary to acute myelitis. This condition is met with more frequently in cases of cerebral softening than of cerebral hæmorrhage.

In some cases there are symptoms that suggest paralysis of the cervical sympathetic (Nothnagel). These are contraction of the pupil, slight drooping of the eyelid (not due to paralysis of the levator, because the eyelid can be raised as high as on the paralysed side), narrowing of the palpebral fissure, retraction of the eyeball, and an increased secretion of tears and of nasal mucus. The pulse on the paralysed side is often smaller than on the other, and sphygmographic tracings are said to show a lessened contractility of the wall of the vessel (Wolff and Eulenberg).

Occasionally the nutrition of the nails is changed, and they become more curved and brittle. Rarely there is an increase in the growth of hair and a thickening of the skin. When hemiplegia comes on in childhood the growth of the limbs is usually retarded, and they never attain the normal size. The difference is more marked in the upper than in the lower limbs, and affects all parts, including the bones; in the scapula it is often very conspicuous.

Disorders of Movement after Hemiplegia.—Besides the rigidity already mentioned the affected limbs sometimes present other forms of spasm,—tremor, rhythmical movements, and especially irregular movements, occasionally quick, more often slow. These occur only when there is some return of voluntary power, not when the paralysis remains absolute. The spasm is especially conspicuous on voluntary movement, although it may also occur, in slighter degree, when the limbs are at rest. All forms are more frequent and more considerable in the arm than in the leg. The spasm does not manifest itself until some months after the onset of the hemiplegia, coming on with the return of voluntary power; once established, it usually persists, although it may lessen somewhat in the course of years.

Simple tremor is not common. It is usually fine, and occurs chiefly during movement, rarely when the limb is at rest. It is confined to the arm. Rhythmical movements are also rare, and likewise occur only in the upper limb. There may be alternate flexion or extension of the fingers or wrist, or pronation and supination of the hand, continuous or only on movement.

The most common form is that in which there is tonic spasm, slowly varying in relative degree in different muscles, and thus causing slow irregular movements, chiefly conspicuous in the hand, and slow irregular inco-ordination. From this feature it may conveniently be termed " mobile spasm." It is commonly conjoined with more or less permanent rigidity, which tends to fix the limb in a certain posture. This fixed rigidity is generally proportioned to the amount of paralysis. If the loss of power is slight, and there is no fixed rigidity, the movements may extend in range. In some cases the movements are quick instead of slow. In rare cases, these movements come on without preceding hemiplegia; such a condition was called by Hammond "athetosis" (=without fixed position). Such primary spasm is similar in characters to that which may succeed hemiplegia, and every gradation is met with between such cases of extensive movement without rigidity and the more frequent cases in which the movements are slighter, and are combined with fixed contracture. The condition has also been termed "spastic hemiplegia" and " post-hemiplegic chorea." This latter term is objectionable, because the condition has nothing to do with chorea, and, except in very rare cases of quick movement, the resemblance to true chorea is remote.

In this condition the upper arm is usually adducted; the elbow-joint is commonly flexed, sometimes extended, and occasionally the extended arm is carried backwards by the spasm and rotated inwards, so that the hand is held against the lumbar region with the palm

FIG. 65.

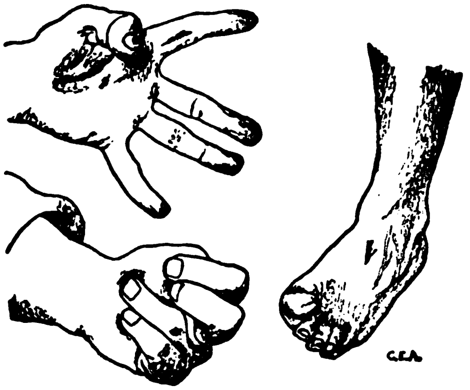

FIG. 66.

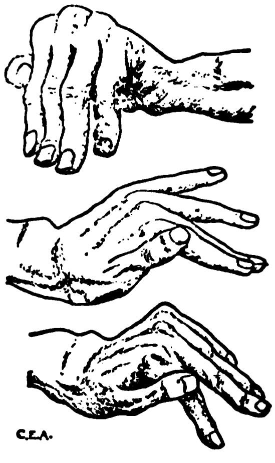

FIG. 65.—Continuous mobile spasm (athetosis) after slight hemiplegia. (Patient aged 24; onset of hemiplegia at 23; of spasm four months later. Previous syphilis.) The hand was in continuous movement between the two positions shown. The foot was habitually inverted, and the great toe often over-extended.

FIG. 66.—Post-hemiplegic mobile spasm. (The hemiplegia came on at 23, probably from embolism, and was severe. Spasm commenced a year after the onset, at the same time as slight return of voluntary power. The figures show some of the postures of the hand six years after the onset, especially the predominant spasm of the interossei.)

turned outwards. In the same patient, the arm may be sometimes extended and sometimes flexed. If there is fixed rigidity, this usually affects chiefly the flexors of the wrist, so that this joint is bent at a right angle. If there is no fixed rigidity, the wrist is sometimes flexed, sometimes over-extended. In the hand (Figs. 65 and 66) the spasm has almost always a peculiar distribution. It affects especially the interossei and lumbricales, which flex the metacarpo-phalangeal and extend the phalangeal joints; sometimes it affects also the long extensor, but never the long flexors of the fingers, which are the especial seat of the ordinary "late rigidity." Hence, the hand is usually in the "interosseal position" (such as the interossei produce), with flexion of the proximal and extension of the middle and distal phalanges, but the amount of spasm varies from time to time in the muscles of the different digits; now one is slowly extended, then another; and the thumb is sometimes pressed against the forefinger, sometimes over-extended. At one time all the fingers may be extended and separated, then one or another is adducted or flexed, the wrist being sometimes flexed and sometimes extended, and the slow irregular changes in position suggest the movements of the tentacles of a cuttle-fish. The constant spasm of the interossei may, after a time, so affect the middle and distal phalangeal articulations that they become over-extended and undergo a subluxation, so that the head of the nearer phalanx is prominent on the palmar aspect of the finger.* The exercise of the muscles by the continuous activity may lead to their overgrowth, so that the affected arm may be larger in circumference than the other, and this even in cases in which growth has been arrested and the limb is shorter than the other. When the hand is at perfect rest, the movements become slighter and often cease, but they are renewed by any attempt at voluntary action, and even by attention. In more severe cases, such as those to which the term "athetosis" is strictly applicable, the movements continue during rest, and may even persist during sleep. On the other hand, in many cases the spontaneous movements are trifling, and close observation may be

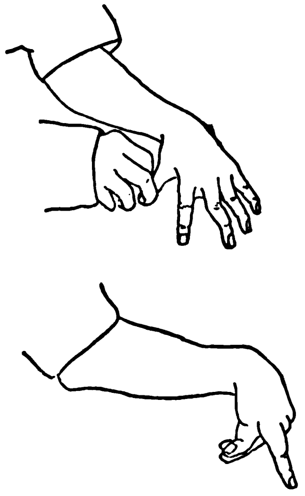

FIG. 67.—Mobile and fixed spasm in hand after hemiplegia in early life. In the upper figure the thumb is held so as to show the over-extension of the middle phalanges of the fingers. As soon as the thumb was released, the hand assumed the position shown in the lower figure.

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* This occurs chiefly when the spasm comes on early in life. In children over-extension of the phalangeal joints can readily be produced, and it is kept up and increased by the spasm.

necessary to detect them. In all cases voluntary movement is irregular and difficult, the spasm excited by the attempt producing a peculiar slow inco-ordination.

The leg is always affected in much slighter degree than the arm. The spasm is extensor, and in the foot tends to cause elevation of the heel and inversion of the foot—talipes equino-varus. Some fixed rigidity in these muscles usually coexists. There is often also a marked over-extension of the great toe. The mobile spasm in the leg is rarely spontaneous, but is usually at once excited by movement, especially by attempts to walk.

The face, in these cases, presents as a rule no spontaneous spasm;* the same spasmodic tendency is nevertheless seen also here, causing a slight movement to be greater in degree on the affected side than on the other, and the result may be of great diagnostic importance. In a slight smile, for instance, the angle of the mouth is drawn outwards more, and often sooner, on this side than on the other, although there may be a manifest deficiency of power on a stronger movement. The slight over-action in the face is often a most valuable diagnostic indication in cases of old infantile hemiplegia, of which little trace may remain elsewhere.

In about half the cases in which this condition follows hemiplegia in the adult, there is impaired sensation on the affected side—hemianæsthesia. But in the cases which date from childhood, sensation is always normal. (A lesion of the brain in childhood seldom, if ever, causes persistent loss of sensibility.)

It has been mentioned that movement intensifies the spasm and is disordered by it, being, in consequence, rendered ataxic or inco-ordinate. In some cases there is no spontaneous spasm, but simply inco-ordination of voluntary movement, varying in degree from mere awkwardness or unsteadiness to wild disorder, and there may be jerking inco-ordination resembling that seen in disseminated sclerosis.

Regarding the nature and position of the disease which causes these disorders of movement we have as yet but little pathological evidence. The symptom is observed after recovery from the paralysis, and hence in patients who, for the most part, live on and pass out of observation. But two etiological facts are of great significance. The first is that these disorders of movement are far more frequent after cerebral softening from vascular occlusion than after cerebral hæmorrhage.† The second is that they follow hemiplegia far more frequently when this comes on in infancy or childhood than when it comes on in adult life. The probable significance of the first fact is that in softening, slight damage to the cerebral tissue is more extensive than the actual destruction, and the spontaneous spasm must be referred to the overaction of grey matter, which is in a state of altered nutrition and function. Hence we can understand the occurrence of this symptom from a lesion which involves extensive slight damage. The significance of the second fact—the frequency with which the condition follows infantile hemiplegia—is probably the greater facility with which the growing and developing nerve-cells recover, and their greater susceptibility to disorder of function when their development is perverted.

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* But cf. a case by Beevor and Horsley, 'Brit. Med. Journ.,' 1890, ii, 1286. Many other anomalous cases have been recorded, chiefly solitary (see Remak, 'Neur. Cent.,' 1888, 392; Blocq and Blum, 'Rev. de Méd.,' 1888, p. 79; Kraft Ebing, 'Wien Kl. Woch.,' 1889, &c.).

† See "Athetosis," &c., 'Med.-Chir. Trans.,' 1876.

Regarding the seat of the disease which gives rise to these symptoms, the facts at present recorded are too few to permit any accurate generalisation. It is, however, probable that a distinction must be drawn between the cases in which the lesion occurs in childhood and in adult life. In the latter, the condition is rare, and in most of the recorded cases the disease has been situated either in or outside the optic thalamus; in several, it involved the posterior part of the internal capsule, a fact which explains the occasional association of hemianæsthesia. The disease of the internal capsule has usually been incomplete, not involving the whole thickness of the capsule, and the lesion has always involved the grey matter of the lenticular nucleus or the caudate nucleus. Since the optic thalamus is not in the motor path, disease limited to this must produce the symptom indirectly by disturbing the function of some motor structures. It is certain, moreover, that the symptom may result from disease which is limited to the cortex.* Regarding the cases that date from childhood we have very few facts. In those that have been examined, the disease has varied much in position and extent. This, and the frequency of its occurrence, suggest that, as mentioned above, the symptom is due to the quality of the lesion (impairment of the nutrition of growing motor nerve-cells) rather than to its site.

The relation between these various forms of post-hemiplegic diseases of movement is shown in the following table.

Post-hemiplegic disorders of movement.†

Quick, clonic spasm, of intermitting type .	Regular (continuous, or on movement) . .	Tremor	Fine. Coarse.
		Certain regular movements, due to interossei, pronators, &c.	
	Irregular (continuous, or on movement) .	Choreoid jerking	Continuous spasm, or incoördination of movement.
Slow, mobile spasm, of remitting type . .	Continuous = "Athetosis."		
	On movement = Slow, cramp-like incoördination	"Spastic contracture" of hemiplegic children.	
Tonic spasm, varying . Fixed rigidity, unvarying	Of interossei conspicuous Of flexor longus digitorum conspicuous = late rigidity.		

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* A conclusive case has been published by Demange, 'Revue de Médecine,' May, 1883, Case ii, p. 375. See also Beevor and Horsley, 'Brit. Med. Journ.,' 1890, ii, 1286.

† From "Athetosis and Post-hemiplegic Disorders of Movement," 'Med.-Chir. Trans.,' 1876, p. 271.

CONVULSIONS.—Convulsions are frequent in organic brain disease. They are produced in two ways: (1) By active irritation, as in meningitis, growing tumours, encephalitis, and in acute cerebral lesions, hæmorrhage or softening. (2) By altered nutrition of grey matter around a stationary lesion; in consequence of the alteration the grey matter possesses deficient stability, and "discharges." By each mechanism convulsions are produced most readily and most frequently when the disease is at the cortex, and the second mechanism is practically confined to cortical lesions. Stationary lesions scarcely ever cause convulsions, unless they are situated in or near the motor convolutions. On the other hand, irritating disease often causes convulsions when it is at a distance from the motor region, both when it is in the cortex itself and in the deeper parts of the brain, even in the pons. Convulsions also result from general increase of intracranial pressure.

The convulsions that result from organic brain disease may be general, and similar to those of idiopathic epilepsy, consisting first of tonic and then of clonic spasm, with sudden loss of consciousness. A full description of their character is unnecessary here, since they are described in the chapter on Epilepsy. The fits are usually of this type when they are due to a diffuse morbid process, as meningitis, or to disease away from the motor region, and also whenever the discharge is intense. But in most cases in which the convulsions are due to focal disease, especially when this is in or near the motor region of the cortex, the convulsion is of a different type, distinct in at least some of the attacks. The discharge commences at the seat of irritation, and spreads thence in the motor region. Hence the onset of the convulsion is deliberate and local; often consciousness is not lost until the cerebral discharge has progressed beyond its point of commencement. The local commencement may be seen by an observer, and often the patient is aware of the commencing fit, and of the manner in which it commences. The local commencement of the convulsion is usually in the side of the face, in the arm, or in the leg. If slight, the convulsion may be limited to the part in which it commences (partial convulsion), and consciousness may not be lost at all. If more severe, the convulsion extends to all parts of the side on which it commences, and consciousness may or may not be lost. If still more severe it may extend to the opposite side, and then consciousness is almost always lost. But there is a further distinction to be made in the mode of affection of the muscles of the two sides. These are involved in different degree and order, according to their unilateral or bilateral use (Broadbent, Hughlings Jackson) and corresponding innervation from one or both hemispheres (see p. 76). We have seen that, in proportion as movements are bilateral, those on the paralysed side escape in hemiplegia. In the same proportion those on the sound side are involved in convulsion. The bilateral representation which permits their escape in one case ensures their involvement in the other. Hence we may have three degrees of extension: (1) In a slight unilateral fit the unilateral muscles of one side are alone involved; the fit affects only the arm and leg. (2) In a more severe fit, in addition to the unilateral muscles, the bilateral muscles of both sides are involved, *e. g.* both sides of the thorax, and sometimes the opposite leg, the arm remaining free. (3) In a still more severe fit the opposite arm is also involved, or the whole of the second side. An intense discharge, of local origin, may spread so rapidly as to appear general from the first. On the other hand, it is important to remember the frequency with which fits due to local disease begin with a sensation, in the limbs or special senses, to which the motor spasm is added. The slightest fits, however, may consist of the sensation only, and this when the discharge occurs in the "motor" region. When there is also spasm, the place at which the sensation begins (not the spasm) points to the seat of instability in the brain.

Yet another distinction is necessary. There are muscles on the two sides, which, together, have a unilateral action. These muscles, as we have seen (p. 78), are innervated from the cerebral hemisphere according to their action, *e. g.* the muscles which move the head and eyes to the right are innervated from the left hemisphere. We have also seen that in unilateral paralysis the movement is lost by an impairment of the muscles on both sides. In a unilateral convulsion the spasm has a corresponding distribution; the head and eyes are usually turned towards the side convulsed, by these contra-lateral muscles, as they may be termed. In a fit which is from the first general, it is common to have the convulsion greater on one side than on the other, with a corresponding deviation of the head and eyes. In a fit in which one side only is at first affected, the other side is often affected later, while the convulsion on the first side is lessening. The passage to the second arm (probably due to the extension of the discharge to the opposite hemisphere) is attended by a corresponding movement of the head and eyes, which, turned at the onset towards the side first affected, are afterwards directed towards the second side, when this becomes involved in spasm.

Convulsions begin locally when the disease irritates a part of the brain in which the centres for the different parts are sufficiently separate, and chiefly when the disease is in or near the cerebral cortex, in the central (motor) convolutions or paracentral lobule. If the centre is destroyed, convulsions rarely begin in the part, which is then permanently paralysed. Hence convulsions occur chiefly from disease which partially damages the centres, or which is in their neighbourhood, and irritates them. When the irritation is in the highest part of these convolutions the convulsion usually begins at the foot; when in the middle, at the hand; when in the lower part, at the face, and then, if the disease is on the left side, is often associated with temporary loss of speech. It is probable that a similar differentiation of spasm may occur in limited disease of the white substance or internal capsule, but it is very uncommon for convulsions to result from disease away from motor nerve-cells. When the convulsion begins in one limb, it may commence by a motion or a sensation, according as the motor or sensory elements lead in the discharge. We do not yet know whether this difference has a localising value. Probably it has not. Disease of the central convolutions may cause convulsions commencing by a sensory aura. We have seen that these convolutions have sensory as well as motor functions. We must at present regard the commencement of the fit by a sensation in the arm, leg, or face, as of the same diagnostic value as the commencement by motor spasm.

In idiopathic epilepsy special sense auræ are not infrequent, but in organic brain disease they are rare, but most important. They signify that the disease is adjacent to the special sense centres in the cortex. Thus I have mentioned elsewhere* a case in which a tumour of the occipital lobe caused a visual aura, and another case in which an auditory aura preceded convulsions due to a tumour which commenced beneath the first temporal convolution.† In a third case a tumour in the middle of the posterior limb of the fissure of Sylvius, invading the first temporal convolution, caused convulsions on the opposite side, which also began with an auditory aura (see the chapter on Intracranial Tumours). The higher the body-heat, the more readily are convulsions produced.‡

The convulsions that attend the onset of a vascular lesion are usually general, but commence on the side which is subsequently paralysed, and they may be confined to this side, especially in the case of surface lesions, and they may continue after hemiplegia has become established; in deeper lesions they usually cease after the onset of the paralysis, and if they continue they usually affect only the unparalysed side. The late post-hemiplegic convulsions usually affect only or chiefly the paralysed side.

Unilateral or local convulsion often leaves behind it transient weakness in the part convulsed, lasting for a few hours and then passing away (post-convulsive paralysis). A severe fit probably produces this weakness by exhausting the nerve-elements (Robertson, Hughlings Jackson). But similar weakness often follows or accompanies very slight fits, especially those in which the first (and sometimes the sole) discharge is sensory, and it is then probably of inhibitory nature.§ If fits succeed each other with great frequency for many days, this paralysis may persist during the brief intervals, and be very considerable in degree, but pass away rapidly after the attacks have ceased.

The great characteristic, therefore, of the convulsions of organic brain disease is their local commencement.‖ A local fit, *e. g.* limited to one limb, and local commencement of a unilateral or general fit, have the same significance. The difference is of degree only. Each shows that the discharge in the brain begins locally, and indicates a local change causing a persistent instability at the spot. But local commencement, while it proves local changes of nutrition, does not prove that the disease is what is called "organic," *i. e.* such as can be detected by the naked eye, or even by the microscope. Such local commencement is sometimes met with in idiopathic epilepsy, but it is very rare in this form, in which the common onset is by a general or a visceral aura, or by initial loss of consciousness. The local commencement, therefore, *suggests* organic disease, just as the visceral aura (as a sensation at the epigastrium), or general convulsion without any warning, *suggests* idiopathic epilepsy. If the local fits are very slight and frequent, the probability that there is organic disease is very great, because there must be an excessive degree of local instability, unlikely to exist in idiopathic epilepsy, in which the morbid state is generally widely distributed through the brain, and the instability is seldom confined to one part. In all cases, therefore, in which fits begin locally, a careful search must be made for any other indication of organic disease. But repeated convulsions from stationary organic disease (in which the discharges spread through the brain) may induce a state like that of idiopathic epilepsy. This has a twofold importance. (1) In diagnosis: minor attacks may occur, especially when the disease is in a developing brain, quite like those of the idiopathic malady. (2) In treatment: the removal of the source of the discharges may not stop them; they may go on, proceeding from the cells secondarily affected. Hence the fits, after infantile hemiplegia, may not be arrested by removal of that part of the cortex that is diseased.

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* 'Epilepsy,' 1881, p. 68.

† Ib., p. 70.

‡ See 'Arch. de Phys.,' 1889.

§ 'Epilepsy,' p. 98.

‖ On account of their careful study by Hughlings Jackson they are often called "Jacksonian."

Tetanic attacks have occasionally, although rarely, been produced by disease of the cerebellum, or by disease that exerts pressure under the tentorium. They may last for hours, and, in the rigidity of the extensors of the spine, bending backwarks of the head, and closure of the jaws, may closely resemble paroxysms of traumatic tetanus. It is not at present known whether they originate from the cerebellum or from the pons. Forced movements of the trunk, as a tendency to rotation, are extremely rare, and their significance is mentioned in the section on Localisation.

Hysteroid convulsions may occur in various organic diseases of the brain, the result of the general disturbance of the cerebral function. Thus I have known them to occur in many cases of tumour of the brain, in meningitis, in hemiplegia (which also caused true epileptiform convulsions), and even at the onset of embolic hemiplegia. Their chief importance is due to the readiness with which they may mislead in diagnosis. The symptoms of hysteria should never prevent a careful search for any indications of organic disease, and do not in any degree lessen the significance of the latter.

Sensory Symptoms.—Loss of Sensation — Hemianæsthesia. — Disease of the brain may impair or destroy sensation, both in the skin, muscles, &c., and also in the organs of special sense. The affection, like that of motion, is commonly unilateral, the side affected being that opposite to the cerebral lesion. It usually depends on damage to the fibres which conduct sensation, and which probably pass through the tegmentum of the pons, and certainly in that of the crus, and in the posterior third of the hinder limb of the internal capsule between the extremities of the optic thalamus and lenticular nucleus, and thence radiate to the central and parietal cortex. Loss of sensation is sometimes due to disease of the cortex itself, but complete hemianæsthesia is rare from such disease, because to produce it a lesion must be very extensive. Some of the sensory fibres are probably connected with the optic thalamus, perhaps also with the lenticular nucleus, but the nature and functional significance of this connection is not yet understood; and it is not probable that the activity of these ganglia influences consciousness, or that their disease causes any loss of sensation. Moreover, outside the posterior portion of the optic thalamus, the visual fibres pass near the posterior extremity of this sensory tract, and then leave it to radiate to the occipital lobes. Near this sensory portion of the capsule also pass fibres from the other nerves of special sense. These special sense tracts have likewise undergone decussation, although that of the optic nerves is incomplete. Hence disease here may cause loss of the special senses, as well as of common sensibility on the opposite side, the affection of vision being hemianopia. To this region, as we have seen (p. 40), the name "sensory cross-way" has been given by Charcot.

Thus in the internal capsule, a common seat of disease, the paths for motion and sensation are separate, but contiguous. It is common for a lesion to damage one much, and the other but little or not at all, and to produce, on the one hand, hemiplegia without, or with only slight, impairment of sensation; and, on the other hand, hemianæsthesia with but little motor weakness, but sometimes with hemianopia. But an extensive lesion may damage both. Sensory fibres seem to reach the motor cortex, and it is common for disease there to cause some impairment of sensation, always most marked in the extremity of the limbs, and in monoplegia confined to the extremity of the limb that is weakened.* Hemianæsthesia may be of purely functional origin, but such cases have the associations described in the chapter on "Hysteria," and are especially characterised by the co-existence of "crossed amblyopia" (*q. v.*) and the absence of hemianopia.

In ascertaining the condition of sensation it is necessary to examine separately the various forms of sensibility. The method of making this examination and the precautions to be taken have been already described (vol. i, p. 8).

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* See the references on p. 20; also Petrina, 'Prager Zeitschr. f. Heilk.,' 1881, ii, No. 5.

Besides the ordinary forms of sensory loss there is one to which much attention has been directed. The patient may be unconscious of the position of a limb, on active or passive movement. In brain disease that causes this defect, the power of active movement is usually lost; but if an extremity, *e.g.* the hand, is placed in a given posture, and the patient is told to imitate this posture with the other hand, he may be so far wrong as to show that he has a very imperfect perception of the posture, and this even when there is no demonstrable impairment of cutaneous sensibility. The hand and fingers should be grasped firmly by the observer, so that the direction of pressure may not *suggest* the posture. The loss is regarded by Munk as a loss of the processes on which "conception of posture" depend, processes that are, in effect, the result of sensory (cutaneous and muscular) impressions. The loss is commonly supposed to be due to cortical disease, but it may, as I have seen, be very distinct when the disease is in the central ganglia. Its actual significance has yet to be established by clinical and pathological observation. This may be said also of the loss of other sensory "conceptions" as distinguished from mere sensations. Thus when tactile sensibility, tested in the ordinary way, seems to be normal, the patient may be unable to recognise the nature of objects in contact with the skin, although they are at once recognised when placed on the unaffected side.

It will be seen, from what has been already said, that loss of cutaneous sensibility may be chiefly on the limbs, and especially on the extremities of the limbs, or it may involve the whole of one side, including the trunk and the head. It is to the latter that the term "hemianæsthesia" is generally applied. The loss often extends up to the middle line, and exists on the mucous membranes as well as on the skin. But it is not always thus complete; it may be more considerable in some parts than in others, and may even be unequally distributed over different regions of the trunk. Nor does it involve equally all forms of sensibility; either touch or pain may be chiefly affected. It is often associated with impairment of the special senses, because, as we have seen, the paths of special and cutaneous sensibility are contiguous, and their cortical centres probably occupy adjacent regions in the outer surface of the cerebral hemisphere. In these cases vision may be impaired either as "hemianopia" or "crossed amblyopia," the significance of which has been mentioned above, and also in the account of the structure and functions of the brain; their characters will be given in the description of affections of sight.

Crossed anæsthesia of limbs and face occurs only in disease of the upper part of the pons, affecting the fibres of the fifth nerve on one side and the path from the limbs on the other. Bilateral anæsthesia, affecting the limbs on both sides, may occur from disease of the pons, but is seldom complete.

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not actually amount to a sense of turning), or a correlated impression that other objects are moving. This involves a lessened perception of the relation of the individual to external objects, *i. e.* a slight obscuration of consciousness. Hence, the corresponding English word "giddiness," and still more the looser term "dizziness," are applied to a slight dulling of consciousness, not amounting to actual loss, although there is no sense of movement, objective or subjective. In pure vertigo there is no actual loss of consciousness, although consciousness may be obscured at the height of an intense attack.

Vertigo is a frequent symptom, apart from organic disease. It results from many causes, and is therefore described more fully in a later part of this volume. Like headache, it does not in itself suggest intra-cranial disease, and derives its significance chiefly from its associations. Of these, vomiting has not the significance which it has when associated with headache. Intense giddiness, whatever its origin, usually causes vomiting, and this does not, therefore, do more than emphasise the fact that the vertigo was severe. This symptom may be caused by disease in almost any part of the brain, and especially by that of the auditory nerve. It is most common in lesions of the cerebellum and of the pons, especially at the side of the pons, involving the middle peduncle of the cerebellum. A lesion here sometimes causes not only a sensation of turning, but an actual rotation. When due to disease elsewhere it occurs chiefly as a symptom of irritation as part of slight "discharges." This association is seen in epilepsy, in which vertigo is frequently the earliest subjective indication of an attack, severe or slight.

Mental Symptoms.—The mental functions of the brain are frequently disturbed in organic disease, and their derangement chiefly depends on disturbance of the cortex. Such disturbance may, however, be produced by disease at a distance, as well as by organic changes in the convolutions themselves. With the much-disputed question of the relation of mind to brain the physician has nothing to do. It is enough for him to recognise that mental manifestations and cerebral activity invariably coincide, and that the character of cerebral processes in some way determines the character of mental processes—in some way determines mental states. The tendency of advanced psychology in the present day is to keep distinct the two series of phenomena. In the study of diseases of the brain we are concerned only with cerebral processes. Unfortunately, however, the chief terms available are those of psychology, and we are obliged, therefore, to speak of mental processes when all that we need to speak of, and are, indeed, justified in speaking of, are cerebral processes. However undesirable such a confusion may be, it is practically unavoidable.

The changes which occur in mental processes, as a result of organic brain disease, consist for the most part in exaltation, perversion, or defect; and these are often combined. These changes may be manifested by loss of consciousness, by delirium, or by chronic mental failure, and are also seen, in a more restricted form, in cerebral affections of speech.

Loss of Consciousness.—The highest general function of the convolutions subserves consciousness, and loss of consciousness is one of the most important and most frequent symptoms of cerebral disease. The terms "conscious" and "consciousness" are, however, used in two senses: first, to signify the subjective knowledge of the occurrence of mental processes; secondly, to designate the outward manifestations of such processes. In medical language the term is chiefly employed in the latter sense. A patient is said to be "unconscious," or to have "lost consciousness," when there is no spontaneous evidence of mental action, and none can be elicited by sensory stimulation. Hence the term "insensible" is often used in the same manner. Another confusion is introduced by the frequent relative use of the words "conscious of" in the sense of cognition or knowing. Thus a delirious patient may be said to be unconscious of what is occurring around him, although he is not said to be unconscious.

Loss of consciousness may occur suddenly or gradually, and may vary in degree, as is, indeed, implied in the statement that its onset may be gradual. The variation may be in the degree of subjective consciousness, or in the external manifestations of consciousness. It is to the latter that the term "partial loss of consciousness" is commonly applied, as, for instance, in the case in which a patient seems to be asleep, but opens his eyes for a moment when spoken to, and then relapses into his former state. Such state of partial loss is sometimes termed "*stupor.*" Complete loss of consciousness, in which a patient cannot be roused, is termed "*coma*" if it is prolonged. In stupor the reflex action in the limbs is preserved, and may be increased, the lower centres being in an over-active state from the deficiency of cerebral control, and the patient swallows automatically liquids placed in the mouth. In coma the reflex action in the limbs may be preserved, but it is often lessened or lost in the more severe degrees, the depressed condition of the highest centres being apparently propagated downwards to the lower. Swallowing is possible only in the less intense degrees of coma. In severe cases muscular tone throughout the body may give place to flaccidity, and myotatic irritability may be lost. The pupils may be widely dilated or small: in stupor they act to light; but in deep coma they are motionless, and the conjunctivæ may be touched without the occurrence of any reflex contraction of the eyelids. When the reflex action of swallowing is lost, the palate generally shares the muscular relaxation, and, moved by the respiratory current of air, causes a peculiar "stertor," which is a familiar indication of the depth of coma. Even the respiratory movements become lessened, apparently in consequence of lowered activity of the respiratory centre; they become shallow, infrequent, and sometimes present other variations, such as the "Cheyne-Stokes rhythm;" these are described elsewhere. The lessened respiratory movements do not clear the air-passages of the secretion which accumulates in the tubes, and finally in the trachea, causing the familiar "rattle" which is popularly and rightly recognised as a frequent harbinger of death.

Loss of consciousness is the result of interference with the highest functions of the brain, those that are the most readily deranged, and it may be produced by almost any one of the morbid processes to which the brain is liable. Chronic diseases cause it chiefly when they affect a considerable area of the cortex, but it may result from sudden lesions in any part of the brain; it is then termed "apoplexy."

Loss of consciousness may be due, however, to other causes than organic disease. It is one of the most common elements of epileptic seizures, and also occurs when the action of the nerve-cells is interfered with by an imperfect supply of blood (as in acute anæmia and syncope), when the renewal of the blood is hindered by mechanical congestion, and also when the blood conveys to the brain toxic material that interferes with the action of the nerve-cells,—material either engendered within the body (as in uræmia), or entering from without (as in various forms of poisoning).

APOPLEXY.—When coma comes on suddenly it is termed "apoplexy." The word means, by its etymology, a *striking off*, and was used by the Greeks, and is still used, to signify sudden abolition of consciousness and power of motion; and this in popular English also is often called *a stroke*.* Cerebral hæmorrhage being the most frequent cause of this condition, "hæmorrhage into the brain" and "apoplexy" came to be used as synonymous expressions. Subsequently the hæmorrhage was itself spoken of as the "apoplexy," the word being thus used to designate the pathological condition causing the symptoms which it before epitomised. Ultimately it was applied to a similar pathological state elsewhere, and thus hæmorrhages into the substance of the lung, the spleen, or the retina were, and still are, termed pulmonary, splenic, or retinal "apoplexies." Such a use of the word is alike needless, inaccurate, and to be avoided.

The chief cause of apoplexy is a sudden organic cerebral lesion, and the most effective is intra-cranial hæmorrhage. It may also result from laceration of the brain, from simple concussion, and also from the sudden arrest of the blood-supply to a part of the brain, whether by a clot brought from a distance (embolism) or formed at the spot (thrombosis). It probably results, in rare cases, from congestion of the brain, although far less frequently than was commonly supposed.* A similar condition may come on in the old without any visible lesion by which it can be caused. This has been termed "simple apoplexy." In the old the brain is shrunken, the convolutions are small, and the spaces between them are occupied by serum. Before this fact was recognised, undue importance was attached to this serum in the cases of "simple apoplexy;" it was thought to be the cause of the symptoms, and the condition was termed "serous apoplexy," a disease that has no real existence, although the word is still sometimes to be heard at inquests and to be seen on certificates of death.

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* We have here a relic of the spiritualistic pathology at one time dominant The act of an unseen executioner seems to be suggested.

The characteristic feature of apoplexy is sudden and prolonged loss of consciousness, not due to failure of the heart's action. The onset may be so sudden that the patient, without warning, falls insensible, as if "struck down" by some unseen hand. Sometimes it is attended with a convulsion. Occasionally it is slow and gradual, occupying many hours in development ("ingravescent apoplexy"). The face may be flushed or pale, but it is rarely very pale. The heart and arteries beat, often with greater force, and sometimes less frequently than normal. The condition is that of coma, already described. In a case of moderate severity the reflex action soon returns, and the patient, after a few hours, presents some indication of returning consciousness, may make some movement, and may open his eyes when spoken to. On the other hand, in severe cases the coma may continue and deepen in intensity, and the patient dies, usually from the interference with breathing described in the account of coma, less commonly from arrest of the action of the heart. Occasionally death occurs at the end of an hour or two, or even less (see "Cerebral Hæmorrhage").

It is not often, however, that there is only this general loss of cerebral function, uniformly distributed, and gradually deepening or passing away. Much more commonly the symptoms of a local cerebral lesion are added to those of apoplexy. Such symptoms—unilateral weakness or convulsion—may precede the loss of consciousness, or they may be recognised during the attack by the indications mentioned on p. 78. As recovery takes place, these symptoms become more and more distinct, and the patient may be found to have lost the use of language.

In *ingravescent apoplexy* the commencement of the cerebral mischief may be marked by symptoms of general shock. There is commonly pain in the head, and there may be other local symptoms. In the course of some hours, or rarely a day or two, consciousness gradually becomes impaired, and coma comes on and deepens. This form of apoplexy, first described by Abercrombie, is usually due to a slowly increasing cerebral hæmorrhage; the local symptoms may indicate its seat.

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* The possibility of congestive apoplexy has been denied on theoretical grounds, but the clinical evidence of its occurrence is strong. See the chapter on "Hyperæmia."

The temperature in cerebral apoplexy is at first lowered, but usually the fall is small, and is succeeded, after twelve to twenty-four hours, by a rise. Its exact course varies considerably according to the cause of the apoplexy, and will be described in the account of the several lesions. An important exception to the initial fall is presented by some cases of a sudden lesion of the pons or medulla, in which the temperature at once begins to rise, and may attain a hyperpyrexial elevation in the course of two or three hours.

The mechanism by which apoplexy is immediately produced has been much debated. The state was formerly ascribed to the pressure exerted by the clot on the rest of the brain, either influencing directly the cerebral tissue, or pressing on and emptying its capillaries (Niemeyer). That such pressure is exerted by a large hæmorrhage is unquestionable. That an increase of the intra-cranial pressure will cause loss of consciousness is also certain. A dog becomes unconscious when there is a pressure on the surface of the brain equal to a column of mercury 130 mm. high. It is highly probable that the intensity of apoplexy is due in part to this cause. But this will not explain the occurrence of the symptom in small hæmorrhages, by which no general pressure is exerted, or not more than is at once relieved by the displacement of the mobile fluid which surrounds the vessels. It will not explain the occurrence of apoplexy in laceration of the brain, or the early loss of consciousness in severe hæmorrhage, in which, as Jaccoud insists, it should, if merely due to pressure, be a late rather than an early symptom. Moreover, it will not explain the apoplexy which results from the sudden closure of a large vessel, a lesion that involves no increase in the intra-cranial pressure. There can be little doubt, from these considerations, and from the cases in which there is no recognisable brain lesion, that the increase of intra-cranial pressure is not the only, and perhaps not the chief causal element. Apart from this the element common to all cases of apoplexy from organic disease is the suddenness of the lesion. Hence it has been generally recognised that one mechanism of sudden apoplexy must be an arrest of function in the cortex ("inhibition" in current phraseology) by the irritation of the sudden lesion. The more suddenly the lesion is produced, the more energetic will be this influence; the more gradual the lesion, the slighter the inhibition. It may thus be absent at the onset of a slow hæmorrhage. The increase in the intra-cranial pressure is effective chiefly in hæmorrhage; it develops gradually, and doubtless intensifies and maintains the coma in severe cases, and is the reason why this is deeper and more prolonged in hæmorrhage than in vascular occlusion. Moreover, pressure is most effective when rapidly developed. Duret has shown that compression slowly applied has to be ten times as great to produce the same effect as when it is sudden. In slow hæmorrhage, pressure may be the chief mechanism, but the amount of hæmorrhage has to be greater; and hence when consciousness is lost in such cases, the patient rapidly passes into a state of great danger.

Although apoplexy is merely a symptom, and not an independent disease of the brain, it is convenient to consider the more important elements of the differential diagnosis of the condition. From the unconsciousness due to cardiac *syncope*, apoplexy is easily distinguished. In the former the heart's action fails, the pulse is weak and imperceptible, the face is very pale, the respiration may be sighing and irregular, reflex action is rarely abolished, and the sphincters are not relaxed.

From the several forms of *toxæmia* the diagnosis is often easy, sometimes extremely difficult, and often most important. It is easy when, on the one hand, the symptoms of apoplexy are preceded or accompanied by those of a local cerebral lesion; or when, on the other hand, the direct or circumstantial evidence of poisoning is clear, or the symptoms of toxæmia unmistakable. Where there are no local symptoms, and where no guiding history is to be obtained, the diagnosis is difficult, but a correct opinion may commonly be formed by an attentive comparison of the symptoms present. There may be, as just observed, indirect evidence of toxæmia; the breath may smell of opium or alcohol; the urine may contain albumen. But albuminuria or a smell of spirits may mislead. Cerebral hæmorrhage often occurs after drinking; spirit is frequently given to a person in a fit. A smell of spirit must therefore only be allowed weight in the absence of any evidence of cerebral mischief. So, too, albumen is always present in the urine in uræmia, but it is also very frequently present in cases of cerebral hæmorrhage. Alone, this evidence of Bright's disease is of little value, unless there is general œdema and the patient is young; then uræmia is more probable than vascular degeneration and cerebral hæmorrhage. But with other symptoms that indicate uræmic poisoning, albuminuria is conclusive. Hence the age of the patient is an important element in the diagnosis. Late life is in favour of brain disease. The history of a fall or blow on the head adds weight to other symptoms of cerebral mischief.

The character of the coma will sometimes guide. In uræmia, and commonly in alcoholism, it is less profound than in cerebral disease. The patient can be roused, at least partially. In apoplexy, in opium poisoning, and in the most intense alcoholic poisoning, the coma may be profound. On the other hand, the patient with cerebral hæmorrhage may sometimes be roused to answer questions. This is still more frequently the case in cerebral softening. Violent struggling is strongly in favour of drink. The mode of onset is important. In apoplexy it is usually sudden; in uræmia it is usually slow. The uræmic patient becomes first drowsy, then comatose. But with convulsions, uræmic coma may come on suddenly. The onset of the coma of opium and alcohol poisoning is also slow. Ingravescent apoplexy is of deliberate onset, but once developed a profound degree of coma is quickly reached.

General convulsions at the onset exclude drunkenness, and also opium poisoning, while they favour uræmia. Apoplexy sometimes commences with a convulsion, and if the convulsion is unilateral, it is strong evidence of brain disease. Rigidity of limbs, or local muscular twitching during the coma, is, if constant in seat, in favour of cerebral mischief; if variable in position, it is in favour of uræmia. Post-epileptic coma is of course preceded by a convulsion, and should be borne in mind.

The state of the pupils is alone of little importance. Great contraction occurs in, and suggests opium poisoning, but it is also present in hæmorrhage into the pons Varolii. The pupils may be normal or dilated in uræmia, in alcoholic or opium poisoning, and in apoplexy. Inequality of pupils, a unilateral symptom, points to brain mischief. The retina should be examined, since the presence of albuminuric retinitis, in the absence of the signs of a localised cerebral lesion, points strongly to uræmia. But in normal sleep the pupils are usually small, and the eyeballs often diverge and may not correspond in position.

Lastly, the temperature should be noted. In uræmia there is persistent uniform depression; in cerebral lesions the initial depression is succeeded by a rise to a point above the normal. But it should be remembered that the temperature may be raised in uræmia by some local inflammation, such as pneumonia. I have known this to cause an error in diagnosis. Hence it is most important that the examination should be thorough.

The differential diagnosis of the *cause* of cerebral apoplexy will be described more fully under the heads of "Cerebral Congestion," "Hæmorrhage," and "Softening."* "Simple apoplexy" cannot be diagnosed during life, since freedom from the symptoms of a local lesion does not afford ground for inferring that there is no such lesion. The prognosis and treatment of the condition are those of the lesions causing it, and are described in the account of those diseases.

DELIRIUM.—Instead of arrest of the manifestation of mental processes these may be perverted, and the perversion may be manifested in speech or action. This condition is termed delirium.

The characteristic of healthy mental processes is that they are in harmony with the actual sensory impressions of the present or with the memory of those of the past. In delirium this concord is lost. Mental processes cease to correspond to reality. There are false ideas —"delusions;" or sensory images arise without sensory impressions —"hallucinations;" or actual sensory impressions excite erroneous sensory images—"illusions," which may or may not seem to the patient to be true.† The condition of delirium is essentially the same as that which constitutes insanity, but the term "delirium" is usually confined to the acute mental derangement that occurs as a consequence of organic brain disease, of pyrexia, of toxæmic conditions, or of inanition. In these conditions the disease of which it is a symptom is otherwise recognisable. The similar mental state which occurs apart from these conditions, and which constitutes the sole evidence of disease, is regarded as "insanity." Delirium is due to brain disease much less frequently than to toxæmic states.

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* A reader who desires a description of the general diagnosis of the cause of apoplexy will find it in my lectures on 'Diagnosis of Diseases of the Brain.'

† These three terms have been variously used by different writers on mental disease, and the resulting confusion has been increased by still greater variety of use in non-medical writings, and of definition in dictionaries. The term "illusion"

Delirium may be "quiet" or "active." In quiet delirium there are delusions and hallucinations (especially of sight) which dominate the patient's ideas. He does not recognise his friends, mistakes inanimate objects for persons, animals, &c., and often talks almost continuously, but usually in a low monotonous voice, with words imperfectly finished, so that considerable attention may be needed to ascertain what is said—a condition which is aptly termed "low, muttering delirium." On the other hand, in "active delirium" the patient tries to act according to his erroneous ideas; he may insist on getting out of bed, going downstairs or out of doors, and may even, under the guidance of some delusion, jump out of a window, or commit suicide in some other way.

Although the general elements of delirium are identical with those of the mental derangement termed insanity, yet certain common features of insanity are rarely seen in delirium. Such are the extreme and persistent emotional depression of melancholia, the exaggeration of idea which characterises some cases of general paralysis of the insane, the outrageous delusions of personal identity met with in the latter and in some cases of chronic insanity; and, lastly, the rhetorical loquacity of acute mania is, to say the least, extremely rare in symptomatic delirium.

Among organic brain diseases that cause this symptom, the most frequent are meningitis, cerebritis, tumours, multiple or extensive softening, and multiple or diffuse degeneration (*e. g.* scleroses).

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is widely used, and is certainly needed, to express erroneous sensory conceptions in which the error depends on the nature of the sense impression itself, and is or may be recognised. Thus the impression of relief in the stereoscopic image is an illusion. It is mainly in this sense that the word has been made by Sully the title of a book ('Illusions,' Internat. Scientific Series). In this sense the word was used by Bacon and by Pope, and the use corresponds with the original meaning of the word, "a deception." If used in connection with morbid mental states it should be confined to false ideas and images, the falseness of which the patient recognises; for these a word is needed, and some writers have thus limited it. "Hallucination" by its derivation is associated with mental disease, and may conveniently be restricted, as was suggested by Esquirol, to "sensations perceived at a time when there are no appropriate external objects to excite them." This also is not inconsistent with old usage. "If vision be abolished, it is called *cæcitas*, or blindness; if depraved and receives its objects erroneously, *hallucination*" (Sir J. Brown, 'Vulgar Errors,' 1646). The use of "delusion" to designate erroneous ideas, and not merely erroneous sensory perceptions, also agrees with the common use of the word, but it is a term of more general application than the others.

There is little in the mere character of the delirium to indicate which of its many causes is at work. In toxic delirium, especially that due to acute alcoholism, there is often conspicuous tremor of the hands and face, and there are usually the visual hallucinations that have furnished a familiar metaphor by which it is popularly known. Chronic alcoholic delirium may present none of these characteristics. The chief difficulty is the distinction between the delirium due to pyrexia, and that symptomatic of organic brain disease. If delirium is the sole nervous symptom, it can only be accepted as evidence of brain disease when the temperature is normal, or at least is not raised more than one or two degrees. Those who have indulged excessively in alcohol may indeed be rendered delirious by a slight degree of pyrexia, and the delirium is then of complex origin; but in the sober, pyrexial delirium scarcely ever occurs unless the temperature exceeds 101°. In the presence, therefore, of sufficient fever to account for delirium, we are not justified in assigning it to a primary disease of the brain unless other symptoms of such disease are present. These may be extremely varied in character, but one of the most important is headache. As Sir William Jenner insisted forty years ago, the headache of fever ceases, as a rule, when the delirium comes on, and if the two co-exist there is probably encephalic disease. Other significant symptoms are persistent vomiting, spasm or rigidity in limbs or neck, inequality of pupil, strabismus or diplopia, and optic neuritis. Optic neuritis now and then succeeds an acute specific fever (especially typhoid and scarlet fevers), but it does not come on during the height of the disease. In other cases the early symptoms of the disease afford important help in estimating the significance of delirium. If delirium distinctly precedes fever, it has evidently the same significance as if it exists alone. If it succeeds a period of initial unconsciousness, or a convulsion, we are justified in assigning to it a primary intra-cranial cause. The only exception is in the case of acute specific diseases in children, in which a convulsion may occur at the onset; but the nature of such cases is usually clear.

Obvious as these rules may seem, it is difficult to exaggerate their practical importance. The neglect of them has been, and still is, a frequent source of error. In many instances, typhoid fever, or catarrhal febricula in children, has been thought to be tubercular meningitis, because undue weight had been assigned to the delirium. Grosser errors, indeed, are not unknown. On the other hand, care should be taken to avoid the opposite mistake. The combination of delirium and pyrexia must not be accepted as evidence of the general nature of the malady until the observer is satisfied that there are no symptoms of nerve disturbance for which a blood-state will not account. The records of every fever hospital will show how often acute brain disease has been mistaken for a specific fever, because the pyrexia has been allowed to obscure the significance of other indications of nerve disturbance.

SIMPLE MENTAL FAILURE is indicated first and chiefly by *defect of memory*, "amnesia" in the widest sense of the word. There is a physical side to memory as to other mental processes. All functional action of nerve elements is attended by molecular changes in them, and never leaves their nutrition in exactly the same condition as before. A state is left, for a time, in which the same functional action occurs more readily; there is a diminution of resistance in the combination of nerve-elements concerned, and this residual disposition is increased by repetition. This constitutes the basis of motor training, in which facility in performing complex actions is acquired by practice. Such actions are learned by a sort of motor memory, which enters but little into the region of consciousness. A similar condition appears to constitute the physical basis of memory, properly so called. During various mental processes, combinations of nerve-cells are in action, which differ according to the diversity of these processes; and the residual disposition to act in the same way, in the same combination and degree, renders possible a perfect revival of the original activity, and on the side of mind a memory of a past image.

Thus there is probably no special faculty of memory, physical or psychical, apart from the general cerebral and intellectual processes. There is, however, or seems to us to be, a peculiar power of voluntary revival of these processes, which is popularly termed "recollection," and for which we have no better scientific name.

The diseases of the brain that affect memory are extremely numerous, and comprehend general impairment of cerebral nutrition, as by an acute fever, severe mental shock, and especially peculiar functional conditions, such as the trance state of hysteria.* It is affected also by organic diseases that damage the nutrition of the cortex, as meningitis, extensive local lesions, hæmorrhage, softening, tumour; these often indirectly impair the function of a more extensive area than is structurally damaged. Loss of memory results also from various degenerative processes, which are for the most part classed as forms of insanity, *e.g.* senile dementia and general paralysis of the insane.

The defect may be seen only in the inability to retain new impressions, although those acquired up to the time of the commencement of the symptoms may be preserved. More frequently the loss is retrospective as well as present, and it is usually the later processes that cannot be revived, the recent, not the remote, past that is forgotten. The loss may extend back for a few days, or for months or years. This is often seen in degenerative diseases of the brain; in senile amnesia, for instance, the events of early life may be vividly remembered, and those of later years be lost, and thus we have the anomaly that "the new perishes, the old endures" (Ribot). A clergyman once came under my notice who had lost all memory of the last twenty years of his life, and maintained, with consistent perversity, that his age, length of parish work, &c., were just twenty years less than they really were. In rare cases, in which lost memory has been regained, recovery has occurred in the reverse order, from the past towards the present. Thus a man, after an accident, had forgotten entirely the events of the three preceding days, and each day was successively remembered until, at last, the accident itself was recalled.* It is said that in the loss of memory the more special acquisitions are first lost, the most general are preserved longest. This is best seen in the case of words, among which proper names are first lost and then nouns, and then names of qualities. In more general loss of memory the order is difficult to trace. Loss of memory is sometimes partial, *i.e.* there is considerable or complete loss of some class of ideas, as, for instance, of a foreign language. These cases, although curious, are of little interest from a medical point of view.

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* Several remarkable cases of complete loss of memory after such attacks have been recorded (see Carpenter, 'Brain,' vol. i).

Another symptom of mental failure, with which indeed the loss of memory is closely connected, is deficient power of attention—that is, the deficient power of excluding from the domain of consciousness all but one subject. This is often mistaken for failure of memory: in hypochondriasis, for instance, there is often thought to be defect of memory when there is only defective use of memory owing to inability to exclude from consciousness the physical sensations, &c., that distress the patient. Closely connected with loss of memory is incoherence of ideas. Instead of the definite sequence of mental processes that we recognise as normal, one mental image excites another by some accidental association (as word-resemblance), which would be, in health, subordinated to logical sequence. Often the connections that determine the morbid sequence are so subtle as to evade detection. This incoherence of idea is portrayed in language. It is conspicuous in delirium, and is also frequent in simple mental failure.

Defects in the moral sense are also common symptoms of mental failure, although not always present. A slight defect may show itself only in a want of sense of propriety. Such defects are relative rather than absolute, and must be measured by their deviation from the normal conduct of the individual. Many actions—such, for instance, as spitting about a room—would be more distinctly pathological in a man of refinement than in an ill-mannered man of the lower classes. Some actions are unequivocal in their significance, as when a patient deliberately micturates on the floor, or exposes himself indecently, without perceiving the impropriety of the action. Urine and stools are often passed into the bed, in cases of brain disease, in consequence of mental failure, when there is no incompetence of the sphincters. This always indicates a considerable depth of mental change, often much greater than appears on the surface. Hence the symptom is of grave significance, and always of such practical importance that its significance cannot be too strongly insisted on.

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* Kömpfen, quoted by Ribot, 'Diseases of Memory,' p. 123.

Another class of actions which result from mental defect are offences against morality and propriety, and these often seem to depend less on want of knowledge of the nature of the action, than on the absence of motive to avoid it. They occur chiefly in the definitely "insane," rather than in the subjects of organic brain disease.

It is important to note that in children with slight mental defect the absence of natural shyness, and the difficulty of producing any deterrent sense of wrong or shame, often afford more conspicuous indications of the mental deficiency than actual lack of intellectual processes. The children are unabashed by the presence of strangers, and are disobedient, mischievous, meddlesome, wantonly injuring their playthings, and even their playmates. The absence of shyness often leads to a pseudo-precocity which fond parents take to be the dawn of genius.

*Affections of Speech.**—Speech is deranged in various ways by disease of the brain. The process of articulation is immediately influenced by a mechanism of nerve-nuclei situated in the pons and medulla, but these are excited to action by centres in the cerebral cortex. Thus there are higher and lower nervous mechanisms; the former is cerebral, the latter bulbar. When there is disease of the lower mechanism, the elements of speech are correct in number and order, but their form is imperfect; there is defective articulation. In disease of the higher mechanism the form of the elements of speech may be correct, but they are defective in number and arrangement. There is, however, one condition in which disease of the cerebrum interferes with articulation,—when the disease involves the structures concerned in both hemispheres. One-sided disease, either of the cortex of the brain or the path from it, may interfere with articulation for a time, but the loss soon passes away, because it is soon compensated by the other hemisphere. The most important movements are bilateral, and, like other bilateral movements, can be excited from either hemisphere. But symmetrical disease of both hemispheres, if it involves the centres or paths for the muscles concerned in speech, may permanently derange or abolish articulation. The condition is termed "pseudo-bulbar paralysis," from the close resemblance which it bears to the effect of disease of the medulla.

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* The most important writings on the subject of defects of speech are those of Trousseau in his 'Clinique médicale' (lect. viii, p. 218, of Bazire's translation); Bastian, 'Med.-Chir. Rev.,' January and April, 1869; Broadbent, 'Med.-Chir. Trans.,' 1872, vol. lv; Bateman, 'On Aphasia,' London, 1870, 2nd edit., 1891 (a thoughtful and comprehensive treatise); Hughlings Jackson, in many places, but especially in 'Brain,' vols. i and ii; Wernicke, 'Der Aphasische Symptomen-Complex,' Breslau, 1874; Kussmaul's article in Ziemssen's 'Cyclopædia' (vol. xiv of the American translation); and, during the last five years, a series of writers too numerous to mention, the most important of whose papers will be found published or abstracted in the 'Neurologisches Centralblatt' and 'Cent. für Nervenheilkunde.'

DEFECTS OF ARTICULATION.—The changes in articulation that result from disease of the medulla and lower part of the pons, vary from a slight imperfection to a loss so complete as to leave only laryngeal tones to represent articulate speech. This defect is termed *anarthria* or *dysarthria*. Considerable loss is usually associated with obvious palsy of the parts concerned in articulation. The most important effects of such definite paralysis are these: in paralysis of the lips the labial consonants, which depend on precise closure of the lips, are defective; *p*, *b*, and *m* are transformed into *f* and *v*. In palsy of the tongue the linguals *l* and *t* are imperfectly pronounced, and tend to become *y* and *th*. Palsy of the palate not only gives an undue nasal resonance to the voice, but impairs the pronunciation of the explosive labials *p* and *b*, because the patency of the nares prevents the needful compression of the air in the mouth; and these explosives are, therefore, transformed into the continuous labial *m*.

In slighter and less defined disease of the bulbar mechanism, terminal or subordinate consonants and syllables are imperfectly pronounced, irrespective of their character, and are often run together or elided in what is termed "confluent" or "elisive" articulation. Sometimes consonants that require delicately adjusted muscular action are omitted, or easier sounds are substituted, and occasionally consonants are unintentionally repeated. The *r* is especially a source of stumbling to these patients; and words or sentences containing this consonant are often employed as tests, *e. g.* "extraordinarily," "artillery." In other cases, again, syllables are unduly separated, in what has been termed "staccato" or "scanning utterance."

CEREBRAL DEFECTS.—The defects of speech that result from disease of the cerebral hemisphere—defects not of articulation, but of the elements of speech—are now known by the name *aphasia*. The word has become current as a general designation for various forms of defect, manifested in the various modes of expression, just as the analogous word "anæmia" is applied to various forms of deficiency of blood.* The word owes its currency to Trousseau, who summoned it from a slumber of two thousand years,† as a substitute for the term

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* The inaccuracy of applying an absolute negation to a partial effect has led to the suggestion of "dysphasia" as a substitute. The term does not, however, seem likely to come into use, a matter for little regret, since the word has not the merit of unimpeachable exactness, and it has an unfortunate resemblance in sound to "dysphagia."

† Trousseau seems to have been unaware of the antiquity of the word. It was apparently "invented" for him, in 1861, by a Greek, M. Chrysaphis (see Trousseau's 'Clinique méd.,' lect. lxi, and the foot-note at p. 218 of Bazire's translation). The history of the more important steps in the discovery of the cerebral relations of speech is briefly this. The faculty of language was first attributed to the frontal lobes by Bouillaud in 1825, and to the left hemisphere, near the island of Reil, by Dax in 1836. The title of Dax's paper is worth preserving. It was "Lesions of the

aphemia, with which Broca had in turn displaced the *alalia* of Lordat. "Aphemia" is sometimes still employed in a restricted application to articulate speech.*

The subject abounds in difficulty, arising partly from its complexity and partly from the uncertainty due to a deficiency of facts, and the obscurity produced by a redundance of theory. Precise knowledge of the effects of local disease on the various relations of language is scanty, and the possible interpretations of the conditions observed during life are numerous. The clinical facts may be and have been variously explained; and it is almost startling to find, when we come to weigh the evidence, how small is the ground on which some theories are based, and how much deficiency of fact a clear diagram may hide. Hence an attempt to state the definite knowledge we possess may seem to be at once meagre in scope and hesitating in assertion. But if the certain knowledge is insufficient to enable us to explain all the complex phenomena, it furnishes a definite starting-place from which to investigate them; and if the obscurity is recognised, we may be saved at least from misplaced confidence. Hypotheses unfortunately cannot be altogether avoided, but it is desirable that they should be simple in nature and restricted in use until more facts have been ascertained.

Speech-processes in the cortex of the brain are sensory and motor. By the former language is received, and by the latter it is uttered. These processes go on chiefly in the left hemisphere in right-handed persons, but in those who are left-handed the processes go on in the right hemisphere, and this even when the patient uses for writing not the left hand but the right. There are, however, similar structures in each hemisphere, and if those on the left side are destroyed, the corresponding parts of the right hemisphere may take on the lost function, and the symptoms of the loss may slowly pass away. The proof of this is that, in several cases of this character, a fresh lesion in the right hemisphere has destroyed the reacquired power, and there has then been no recovery. It is probable, moreover, as we shall presently see, that the right hemisphere normally takes some part in speech-processes, and that the part it takes varies in different persons and at different periods of life. Loss of speech from disease of the left hemisphere is, as a rule, quickly recovered from in children by the compensatory use of the right hemisphere; a similar recovery takes place in some adults more readily than in others, and in some it does not take place at all. If such compensation occurs—and the occurrence is unquestionable—it is evident that very soon after the left

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Left Half of the Brain coinciding with Loss of Memory of the Signs of Thought." The function was further limited to the third frontal by Broca in 1861. The localisation of the auditory centre in the first temporal is due to Meynert and Wernicke, and to the latter belongs also the credit of the localisation of word-deafness to this region of the left hemisphere.

* "Aphemia" was rejected by Trousseau and by Broca himself, at the instance of Littré, because its Greek significance is not "speechlessness" but "infamy."

hemisphere has lost some of its function, the right hemisphere must begin to co-operate with it in effecting the speech that quickly returns. Such co-operation, admitted when the damage is great, probably occurs also when the damage is partial. It is evident that we have here an element of great uncertainty. If the actual lesion is not known, we cannot be sure to what extent the speech-processes that return, and therefore the disorder they present, are to be ascribed to the left hemisphere or to both hemispheres; and we must incur some error, and it may be great error, if we fix our attention exclusively on the left hemisphere. Examples of this will be manifest as we proceed.

The sensory relations of speech are with hearing and sight, and also, in blind persons, with touch. The relation to the sense of hearing is the most important; it is the first speech relation to be developed in the child, who can understand many words before he can utter one; it seems to lead in the subjective revival of speech-processes which we call "recollecting words." The part of the brain concerned in the auditory perception of words is the posterior half of the first temporal convolution on the left side. It is apparently the auditory centre itself, at least structures at present inseparable from this centre. If this region is diseased on either side, there is often transient deafness in the opposite ear (see p. 24); and if on the left side, there is usually persistent inability to understand the meaning of words, although they are still heard as sounds. This condition causes considerable derangement of speech. It was first described by Dr. Bastian, and accurately referred by him to destruction of the "auditory perceptive-centre." Wernicke, to whom belongs the credit of discovering the precise seat of the disease that causes the symptom,* proposed for the disturbance of speech the term *sensory aphasia*, and Kussmaul afterwards called the loss *word-deafness*. The loss is now generally known by the latter term, and the former is applied to the general disorder of the use of words that results. The dependence of this symptom on disease of the first temporal convolution is well established. This part was diseased in every one of seventeen cases collected by Sepelli.†

The part of the brain concerned in the perception of *visual* word-symbols seems to be in the lower and hinder part of the parietal lobe, also on the left side of the brain, apparently including the region rather vaguely described as that of the "angular gyrus." Disease of this part has caused inability to read even the simplest word, a condition that has been termed "word-blindness" (see p. 5). Of the cases collected by Sepelli, in each one in which word-blindness co-existed with word-deafness the disease extended into this part of the parietal lobe. This region, as we have seen (p. 22), is probably the seat of the "higher visual centre," and transient "mind-blindness" has been caused by its disease, *i. e.* an inability to recognise not only words, but objects also. Such mind-blindness has also occurred in a few cases as a persistent symptom, but we are not sure whether it then depends on a unilateral lesion or on bilateral symmetrical disease (see "Affections of Vision"). The extent and degree to which visual word-processes take part in the phenomena of ordinary speech probably vary much, according to the education of the individual, among other conditions. Their reality is readily evident on self-observation, and is demonstrated only too clearly by cases in which the loss of the auditory and motor word-processes (physical memories) leaves the patient unduly dependent on those connected with sight, or conversely, in which the loss of the last alone has given rise to a peculiar train of disturbances of speech. These are rare, but some instances have been lately studied and described under the term "*visual aphasia*."* The symptoms of this condition, supposed to be produced especially by disease of the cortical visual centre or of the fibres connecting it with the motor speech-centre, are these:—defect in the right halves of the visual field, inability to read (alexia) or write (agraphia), and inability to name objects after looking at them, although they can be named if the patient is allowed to touch and handle them. But they will no doubt be influenced by the degree of education of the patient, and also by the nature of the speech-process for which they are employed (see also p. 119). The pure visual difficulty must be distinguished from that of writing with which it may be combined. Further, the crossed amblyopia, which results from extensive disease in this part, has not often been recognised in cases of impairment of visual word-processes. The speech-defect interferes with the detection of the visual loss, and it may be that word-processes are related to only a part of the region that has to do with vision. It must, however, be noted—and the fact very much increases the difficulty of the subject—that inability to read does not prove disease of this part in the same way as inability to understand heard words proves disease of the first temporal. The mechanism of reading is complex, and, as we shall see, it may be deranged indirectly.

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* 'Die Apha sische Symptomen-Complex,' 1874, a work of remarkable ability.

† Sepelli, 'Rivista Sper. di Fren.,' 1884, p. 94 (see also Amidon, 'New York Med. Journal,' January, 1885).

Motor word-processes take place in the posterior part of the third frontal convolution, and in the adjacent part of the ascending frontal. The latter contains the cortical centre for the movement of the tongue, lips and larynx generally, to which the motor speech-centre thus corresponds. The path from it passes through the white substance of the hemisphere to the internal capsule at the junction of its two parts, in front of the fibres of the pyramidal tract, and it lies to the inner side of these fibres in the crus cerebri. It is commonly thought that the path from the speech-centre is distinct from that for the tongue and lips, but the evidence of this is insufficient. It is more probable that the speech-path is that for the simple movements of the parts concerned, although the cortical centre is more extensive. Whether the island of Reil forms part of the speech-centre is still uncertain; speech-defect from disease limited to it has not yet been observed, but its position between the motor and auditory centres, and the way in which fibres between the two pass by it, have led Wernicke and others to regard it as part of the speech-region. All the cases adduced as proof of the dependence of aphasia on disease of the island of Reil are inconclusive, either because the lesion was not limited to it, or because the duration of the case was too short to permit indirect symptoms to pass away.*

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* See especially Freund ('Arch. f. Psy.,' 1889, xx, 1, 2).

Destruction of the motor speech-region on the left side causes total or almost total loss of voluntary speech. Such loss has resulted from a lesion in the third frontal no larger than a hazel-nut.† If the loss is old or partial, words are often uttered but are wrongly formed. Such defect has been termed *ataxic aphasia*,‡ but this, as a distinctive term, is inexact, since a very similar disorder of words may arise from pure word-deafness. Hence it is far better, with Wernicke, to term the effect of disease of the motor speech-region "*motor aphasia*." Its precise characters will be presently described.

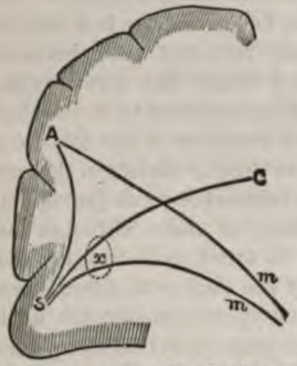

FIG. 68.—Diagram of probable course of fibres from motor speech-centre. A, hand-centre in the middle of the ascending frontal convolution; A *m*, fibres from this to internal capsule; S, motor speech-centre; S C, fibres from this to the corpus callosum; and S *m*, to the internal capsule. S A, fibres from speech-centre to hand-centre. A lesion at *m m* will cause only transient aphasia, the speech-processes being able to pass by S C to the corpus callosum and opposite speech-region; a small lesion at *x* would cause permanent aphasia, since it involves both the fibres to the corpus callosum and internal capsule, but would not abolish expression by writing, the fibres S A (connecting the speech- and hand-centres) escaping.

Motor aphasia may be produced by disease of the conducting path from the cortex, as well as by the disease of the cortex itself.§ The loss from disease of the path is only permanent when the lesion is just beneath the cortex. If the lesion is near, or in, the internal capsule, the loss passes away in the course of a few weeks. The explanation of the difference is probably this: if the fibres (S C, Fig. 68) from the left speech-centre to the corpus callosum are intact, the speech-processes arranged on the left side can find expression through the corresponding centre on the right side; if the lesion is just beneath the cortex (as at *x*, Fig. 68), these callosal fibres are interrupted, as well as those to the internal capsule (S *m*), and the loss is as permanent as if the centre were destroyed. In the former case, a second lesion in the motor path of the right hemisphere, even in the internal capsule, may cause permanent aphasia.*

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* Thus, in a case published by Dejerine, as evidence that motor aphasia may be due to an insular lesion ('Revue de Méd.,' 1885, p. 174), the patient lived for a few days only, and the lower part of the central convolutions was likewise destroyed. (Compare also the case mentioned in the note on p. 116.)

† Rosenstein, 'Berlin. klin. Wochensch.,' 1868, p. 182.

‡ First by Sanders, 'Edin. Med. Journ.,' March, 1866; soon afterwards by Ogle.

§ Raymond and Artaud collected twenty-three cases of aphasia from disease of the white substance ('Gaz. méd. de Paris,' 1883, No. 47).

In the act of writing, the speech-processes, arranged in the motor centre, pass to the centre for the hand in the middle of the ascending convolution, and thence the nerve-impulses pass by the pyramidal tracts to the cord. The evidence of this is that disease of the motor speech-centre abolishes the power of writing (*agraphia*), even when the hand-centre is intact and the movements of the arm are unimpaired.† The same result follows an isolating lesion just beneath the cortical speech-centre, and hence the path to the arm-centre must be by the "associating fibres" of the subjacent white substance, and not through the grey matter of the cortex. But it is conceivable that a subcortical lesion may be so placed as to interrupt the paths to the internal capsule and to the opposite hemisphere, and not that to the arm-centre (see Fig. 68). In such a case there would be permanent loss of uttered speech without loss of the power of writing. Such a condition has actually been observed.‡

Disease limited to the motor speech-region sometimes abolishes the power of reading (*alexia*),§ sometimes it does not. The difference depends in part on the degree to which motor speech-processes are concerned in the comprehension of visual speech symbols. In persons accustomed to reading, the intellectual images may perhaps be aroused directly by the visual processes, without the intervention of the motor processes. In those who are imperfectly educated, the motor processes are essential; the lips may even be seen to move during the process of reading. It must be remembered that children learn to read aloud words they do not understand; indeed, the early effort of a child is merely to pronounce the seen words.‖

In addition to the two leading varieties of aphasia, the motor and sensory, there is another in which the patient, without loss of the motor processes, and without word-deafness, has a difficulty in recalling words, and uses wrong words. This has been termed "amnesic aphasia," or "loss of memory for words." It has been referred by Wernicke to an interruption of the path between the motor and sensory centres, by disease of the island of Reil, and he has termed it "conduction-aphasia" ("Leitungsaphasia").[*] He assumes that the interruption prevents the direct action of the sensory on the motor centre which is necessary for its guidance.[†] The defect that accompanies "word-blindness" has been termed "visual aphasia." As a last variety, "total aphasia" has been described—the form in which both auditory and motor centres are destroyed, and all use of words is lost.

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* This appears to have been the case in an instance recorded by Luys, 'L'Encephale,' 1885, No. 3.

† In Rosenstein's case ('Berlin. klin. Wochenschr.,' 1868, p. 182), in which aphasia resulted from a hæmorrhage the size of a hazel-nut in the third frontal, the power of writing was lost as well as speech. The impairment of writing in motor aphasia is a matter of daily clinical observation.

‡ Wernicke, 'Neurol. Cent.,' 1886, p. 124.

§ Alexia, be it remembered, is the loss of the power of understanding written or printed words, not merely a loss of the power of reading aloud.

‖ At the same time it is doubtful whether the observed differences in the influence of motor aphasia on the power of reading can be altogether explained by the degree of education and of practice in reading. We need more facts regarding the pathological lesions in these cases.

A variety of paroxysmal recurring difficulty in reading has been called *dyslexia,* and compared with the "intermitting lameness" mentioned in vol. i, p. 297. The difficulty is considerable or absolute, comes on suddenly, and passes off gradually, being at first readily reproduced, although afterwards the patient is free until another attack. Most subjects have been in advanced life. It is important because usually the harbinger of grave and progressive disease, generally due to arterial degeneration.[‡]

Motor Aphasia.—In a case of motor aphasia from destruction of the motor speech-region the patient is at first speechless, and even expressions by signs may be impaired. He may shake his head when he means "yes." Before long, expression by gestures becomes accurate, and the patient regains the power of uttering a few simple words, as "yes" or "no,"[§] and often other words are at times uttered under the influence of some emotion, or some word may be repeated on every attempt to speak. The patient is able to understand whatever is said to him, but cannot repeat it. He cannot, of course, read aloud, and he is generally unable to understand seen words, such, for instance, as a written or printed direction to perform some simple action. He cannot write, even if his hand is unaffected. He may be able to copy, and even to translate printed letters into written characters. If the right hand is paralysed, the patient may try to write with the left hand and may try to form words with separate letters, but the latter is a rather severe test because it is an unfamiliar process. Processes of thought are little interfered with, and the patient is perfectly aware of his errors in utterance. The use of figures is usually interfered with as well as the use of words, but to this rule curious exceptions are met with, the significance of which we are not yet able to understand. Thus, in one case of considerable motor aphasia recorded by Volland, a high degree of arithmetical ability was preserved.*

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[*] That defects in speech may be due to interruption of the "set of efferent fibres emerging from the auditory perceptive centre having to do with the incitation to the muscular acts of speech" was suggested by Dr. Bastian in 1869 (loc. cit.). Apart from the question of accuracy, the term "conduction-aphasia" does not very aptly express the assumption that conduction is arrested.

[†] There is at present no actual proof of the truth of this assumption. Lichtheim has published a case ('Deut. Arch. f. klin. Med.,' Bd. xxxvi, Case 1) which he describes as an "undoubted example of this form of aphasia," but the lesion involved, besides the island of Reil, 1½ cm. of the third frontal and part of the middle of the first temporal. (See also note on p. 114.)

[‡] See Berlin, 'Neurol. Cent.,' 1883, p. 296, and 1886, p. 355; also Nieden, ibid., 1887, p. 105, and Pick, ibid., 1891, p. 130.

[§] I have only seen one case in which a patient, nine months after the onset, was absolutely wordless. There was considerable word-deafness as well as absolute motor aphasia. Expression by signs was extensive.

In some cases this condition persists unchanged for years. More often there is, after some months, a slow recovery; first one word and then another is uttered, and considerable power of speech may be ultimately regained by the "re-education" of the right hemisphere. In other cases, however, in which the disease is partial, the loss of speech is found to be incomplete when the immediate effect of the lesion has passed away, but many mistakes are made in the forms of words and in their use.

In the conditions thus sketched there are two elements: (1) loss of speech; (2) retention of speech, or at least of utterance of words, with more or less error. The loss is due to the lesion; the retention and error are due to the imperfect action of the remaining motor speech-centres, probably on both sides of the brain if the disease is partial, or on the right only if it is complete. It is obvious that when there is complete destruction of the left motor speech-centre, the utterance that remains must be by the right hemisphere. This remaining power of expression may be of several kinds:—(1) Expression by gesture, which may be at first impaired but is soon regained. (2) Some general propositional utterance, as "yes" and "no," which may be at first used wrongly. (3) Some special utterance, which recurs on all attempts to speak, and may be a word or words, or an unmeaning combination of syllables. (4) Combinations of words which express some strong emotion, and not the proposition to which their form corresponds, such as an oath—"dead propositions," Hughlings Jackson has aptly called them. (5) Words as the vehicles for tone in singing. A patient who can only say "yes" and "no" may sing a song, and in doing so may utter every word of the song although he is quite unable to speak it.† Often words can be uttered in a half-automatic manner, *e. g.* in combination with other words or under emotion, and not by a deliberate voluntary effort. Thus a patient with slight aphasia was told to say "no," and after one or two ineffectual attempts said, "I can't say no, sir." Thus likewise I have known a patient to be unable to put out his tongue when told, although he automatically put it out and licked his lips to facilitate the process, and afterwards was still unable to protrude it by a deliberate effort. An oath uttered in anger cannot be repeated without the emotion.

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* 'Munch. med. Wochensch.,' 1886, and 'Neurol. Centralb.,' p. 274. The patient was a boy aged fifteen. After a fall on the head there was complete motor aphasia, only the recurring utterance "Anna" persisting. Recovery was very slow, and the defect had not entirely disappeared six years later. From the first the boy could add, subtract, and multiply, and at school he progressed steadily in arithmetic.

† See 'Lancet,' 1875, vol. ii, p. 794. For an interesting discussion of this condition see paper by Knoblauch in 'Brain,' 1891.

The "recurring utterances" are very curious. They seem to be either the words the patient was about to utter when taken ill (Hughlings Jackson), or more probably the last words actually uttered. A woman who told a cabman to drive her to "Mrs. Waters," and became speechless a few minutes afterwards, had the recurring utterance, "Missis." The recurring utterance of a girl, seized when riding on a donkey, was "gee gee" (Jackson); that of a signalman, taken ill at his post, was "Come on to me;" that of a librarian was "List complete" (Russell). The phenomena of recurring utterances, which must be effected by the right hemisphere, seem to show that this hemisphere takes some part in normal speech. Fresh processes for words cannot be energised through the will, but the residual disposition of those last energised voluntarily, leads to their excitation by an attempt to speak. As Hughlings Jackson has insisted, the loss of speech from disease of the left motor region is essentially a loss of *voluntary* speech. When speech is slowly regained by the right hemisphere, many of the errors in utterance may be traced to the defective voluntary influence, and to a tendency to the re-energising of nerve-processes recently in activity. Consonants are repeated instead of the proper consonant being uttered, and those of a recurring utterance may crop up in wrong places. When the patient whose recurring utterance was "Missis" acquired more speech, the "s" was constantly cropping up in other words. Ultimately almost perfect recovery may occur, and there may remain only slight and occasional errors in the form of words, with a difficulty in finding the word desired, and a tendency to use wrong words.

In motor aphasia the inability to write is usually even greater than the inability to speak. In writing, speech symbols are again symbolised, and in the double symbolism there is more room for error. The patient may be unable to form a single letter, or letters may be formed but combined wrongly, or there may be merely mistakes of letters in words that are otherwise correct. The patient can often sign his own name—which is almost an automatic performance—when he cannot express a proposition in writing. As an instance of this, and also of the fact that errors in writing are greater than in speech, may be given the case of a man who, after signing his name, *James Slim*, correctly, was told to write down "how he came to the hospital," and wrote *cgng kgig kiyan*, but in answer to the same question said, "Rail, clab," and when asked "How else?" replied "No way." The tendency to *repetition*, to the re-energising of processes recently in activity, causes many of the errors in writing as well as in speech, and is seen in the above sentence. *Glag* for "glad" is another instance. Other mistakes in written letters are due to a similarity of the muscular (and therefore nervous) action in pronouncing them, as the substitution of *l* for *p* and *f* for *v* (observed errors)—a proof, if proof were needed, that the written words are first energised in the speech-centre.

Loss of the power of understanding seen words, that is loss of the power of reading—"alexia"—often, but not always, accompanies motor aphasia. This has been already mentioned, and it has been pointed out that the difference probably depends on the extent to which the patient is dependent on the motor processes for the comprehension of the visual symbols. In some persons the latter arouse the nerve-processes for "ideas" directly, in others only by the energising of the motor structures. In the former case even disease of both temporal and frontal speech-centres may not destroy the power of reading, although, of course, the patient cannot read aloud.[*]

Sensory Aphasia.—In word-deafness, due always to disease of the first temporal convolution, heard words are not understood. This is best tested by letting the patient perform some simple action which is not anticipated. Word-deafness is seldom absolute, and an expected sentence, such as "Put your tongue out" from a doctor, is understood when an unexpected sentence is not. Expectation involves lowering of resistance in a certain nerve combination, and then perhaps the right hemisphere may suffice. Word-deafness always causes considerable derangement of speech. This depends on the fact that the subjective revival of word-processes takes place chiefly in the auditory centre. All the residual dispositions—motor, auditory, visual—constitute physical memories for word-processes, but as auditory word-processes are the first to be developed in the child, so they continue the most important through life. If they are lost the patient is thrown on his motor memory, and this often misleads.[†] Hence there is often error in the words used and in their form. But it is especially the voluntary revival of words that is lost, and words may still be revived automatically, just as it is voluntary utterance that is lost in motor aphasia, and words may still be uttered automatically, as in singing a song. Hence these patients have often an extensive use of words, especially general words, which they utter promptly, and in an automatic manner, but they have difficulty in the deliberate revival of special terms. "If I have to say a thing I must say it at once, or I cannot say it at all," one patient observed. It is probable that the automatic revival of words in disease of the left hemisphere is subserved by the centres on the right side. It is certain that slow recovery of the power of comprehension of spoken words is by the "re-education" of the right hemisphere, since such regained power has been again lost in right-sided disease. The readiness with which such recovery takes place seems to vary much in different persons, as is the case also in motor aphasia. In some there is no recovery.* As a rule, however, recovery occurs more readily than in motor loss.

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[*] As in a case reported by Kahler, 'Cent. f. Nerv.,' 1885, p. 393. The statement in the text is only a probable explanation. We need more facts as to the influence of definite lesions on reading.

[†] In educated persons the visual memory may aid in the revival, and probably develops with the demand upon it. An accomplished man recovered slowly from complete word-deafness (with persistent hemiplegia). He said that if he had a difficulty in realising the meaning of a word, he would repeat it over and over, and at last seemed to see the letters of the word, and its meaning flashed upon him. This is an interesting instance of the way in which one centre may reinforce another. A different example of similar help was presented by a patient who could write, but could only understand a word seen if he traced the letters with his finger in the air (Charcot, 'Leçons,' t. iii, p. 161, 1887). The same case has been recorded by Herczel. So, too, a patient of Wernicke's was able to read writing only by tracing over the letters ('Gehirnkr.,' i, p. 338).

The disorder of speech in word-deafness varies much in different cases. As a rule, considerable use of words remains, but chiefly in half-automatic spontaneous speech. Parts of speech in more general use, prepositions, adjectives, verbs, &c., are readily uttered, but there is great difficulty in more special words, such as nouns. The patient often employs a circumlocution of more general words instead of the special word that he cannot revive. Instead of the word "knife" he may say, "something to cut with." In extreme cases there may be much error in the forms of words, and these errors are often distinctly due to the inadequacy of the motor memory on which the patient has to rely. Sometimes, indeed, there is as much formal disorder and "ataxy" of speech as in motor aphasia—one of the considerations that militate against the use of this term as a designation for the motor variety. In very rare cases extensive disease of the sensory region has caused an almost absolute loss of expression by words, greater indeed than can be altogether explained, and such as to resemble the loss in disease of the motor region.† The effect of disease of both temporal lobes is, of course, to preclude recovery, and great derangement of speech results. In one case of complete deafness from cerebral disease, in which such a double lesion probably existed, speech was almost unintelligible.‡

Word-deafness renders the patient unaware of his errors—a striking difference from the motor form, in which the patient at once recognises his mistakes, and is annoyed at them. In absolute word-deafness the patient is unable to repeat words uttered to him, but in partial loss he is able to repeat, often with some formal errors.

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* As in a case recorded by Rosental, in which word-deafness remained absolute until the death of the patient, two and a half years after the onset ('Gazeta lekarska,' 1883, and Virchow's 'Jahresb.,' 1883, ii, p. 85).

† Thus, in a case recorded by D'Heilly ('Prog. Méd.,' 1883, No. 2), arterial thrombosis in the course of phthisis caused softening of the left first and second temporal convolutions and of part of the angular gyrus, without implicating the motor speech-region; the patient, during the nine weeks she lived, only uttered the words "parceque," "le plan," and "oui, monsieur." She understood no words, but comprehended signs readily. She had lost the power of reading and writing. In a case of Charcot's, described by Trousseau, in which the lesion involved only the first and second temporal convolutions and the posterior half of the island of Reil, the patient uttered only "ta-ta-ta" during eight months.

‡ Wernicke and Friedländer, 'Fortschritte d. Med.,' 1883.

The patient is also unable to read in many cases of word-deafness, but it is probable that this is the result of the auditory loss, or is due to damage to the visual centre, which is supplied by the same artery as the auditory centre, and often suffers in the same lesion. Inability to read, as an isolated symptom, is probably always due to a lesion of the supra-marginal and angular convolutions.* It is not often that there is actual mind-blindness, an inability to recognise not only words but any object until some other sense than that of sight is employed (see "Affections of Vision"). In some cases the patient has been able to read aloud correctly, but not to understand what he read. The effect on writing is similar to the effect on speech, but greater, owing to the double symbolism and the extension of error thus produced.

We have seen that in partial word-deafness, partial from the beginning or partial by recovery, a condition exists in which there is great difficulty in the voluntary revival of words, especially of the more special words, such as nouns. This is "verbal amnesia," or "amnesic aphasia."† As already stated, it sometimes constitutes the sole defect from the beginning, and is then commonly regarded as a distinct variety of aphasia, and has been ascribed to interruption of the conducting path between the auditory and motor centres (see above, p. 116). Whether the condition is actually thus produced, and whether a simple interruption of this path can produce this effect, remain to be proved. The condition is often conjoined with slight word-deafness, and then is probably due to a partial lesion of the auditory word-centre. It may not only be a residual condition left by partial recovery from a considerable degree of word-deafness, but, as we have seen (p. 118), it may be left by recovery from complete and pure motor aphasia. The fact that a typical "amnesic aphasia" may be merely a residual condition left by a pure motor aphasia is very important. It is proved by several recorded cases in which an autopsy was obtained.‡ Another important fact is that the loss follows a regular law from the special to the general. These facts suggest that the essential condition for the production of this defect is that the speech-processes go on in structures relatively incompetent. In some cases the structures concerned may be those of the right hemisphere, imperfectly re-educated. This form can only, therefore, be admitted as a pathological variety of aphasia with some reservation, until more evidence is obtained regarding the lesion that causes it as a primary and independent defect.

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* As in a case recorded by Henschen, 'Neurol. Cent.,' 1886, p. 464.

† The use of the word amnesia in this restricted sense is open to the objection pointed out by Hughlings Jackson and Kussmaul, that there is more than one memory for words (see p. 119).

‡ One is recorded by Batty Tuke and Fraser ('Journ. of Mental Science,' April, 1872); another by Sanders ('Edin. Med. Journ.,' 1866, p. 811).

Inability to read, *alexia*, as we have seen, may have more than one mode of origin. It may be part of a pure motor aphasia in those in whom visual word-processes can only arouse mental images through the agency of the motor speech-centre. It seems strange that the motor process should directly arouse the nerve-processes for the mental conception,* but there is no evidence that the auditory processes intervene. On the contrary, the loss of these processes does not necessarily abolish the power of reading, as would certainly be the case if the motor centre acted on consciousness only through the auditory centre.† In other cases of motor aphasia the power of reading is unimpaired, presumably because the visual processes arouse directly those for "ideas." Without motor aphasia, loss of the power of reading seems to be due only to disease of the posterior-inferior region of the parietal lobe, the region of the higher visual centre. We have seen that in all the fatal cases of word-deafness in which the power of reading was lost, the disease was found to extend into this region. Hence we are justified in assuming that when alexia exists as an isolated condition, the disease is in this part. This isolated loss of the power of comprehending visual word-symbols has been termed by Kussmaul "word-blindness." It may or may not be accompanied by "mind-blindness," which necessarily involves alexia. (See also p. 116, *visual aphasia.*) Word-blindness is indeed partial mind-blindness. In this condition there may be ability to speak and to write, but the patient writes, as one of Charcot's patients expressed it, "as if with the eyes closed," and this patient did actually write as well when his eyes were closed as when they were open.‡ Moreover he could understand written words if his hand were made to trace the letters in passive movement. Such loss of visual word-symbols has been met with in a few cases of hemianopia; it is not a necessary consequence of hemianopia even when this is of cortical origin, and probably indicates that the lesion extends beyond the limits of the occipital lobe into the parietal cortex.§ The lesion causing alexia is always on the left side of the brain in right-handed persons, on the right side in left-handed persons.* The varying character of the symptoms due to disease of this part of the parietal lobe may be due to the fact that its blood-supply is often derived from two different arterial systems, and the area supplied by each seems to be subject to considerable variations. Hence partial disease is the rule, complete destruction the rare exception.

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* *I.e.* it may seem strange that a centrifugal process should have an influence which seems to be centripetal; but a similar influence attends other motor processes, *e.g.* our estimate of position by the movements of the ocular muscles (see "Diseases of the Third Nerves"). All motor processes seem to influence consciousness, whether as a "sensation" or only as a vague conception or sense of the effect.

† In this connection it is instructive to note that even the motor processes for writing may assist in arousing the mental images, as in the case mentioned in the note on p. 119. Wernicke and Kussmaul assume that the auditory centre intervenes, and that the uneducated man, who can only read by the help of his motor centre, "has to read aloud to himself" in order to understand—an assumption opposed to facts.

‡ Charcot, 'Leçons,' tome iii, p. 160.

§ A lesion of the white substance involving the optic radiation may also involve the parietal cortex.

Inability to write, *agraphia*, as we have seen, results from destruction of the motor speech-centre, and is then usually absolute, even when the right hand is not paralysed. Spontaneous writing, and writing from dictation, are alike abolished, and hence it would seem that there is no direct path from the auditory word-centre to the arm-centre, but that the word-processes must first be energised as for articulate speech. But the patient is able to copy, usually only in the characters that are before him; sometimes, however, by a translation of printed into written characters. It is certain that, very often, the words so copied are not understood. In word-deafness, as we have just seen, the ability to write persists, but not to write from dictation. In word-blindness, the patient can write, spontaneously or from dictation, but cannot copy. The difficulty in writing, as before stated, is often greater than in other forms of expression, but it is very seldom that it so far preponderates as to resemble isolated agraphia. The possibility of isolated agraphia has been doubted, and is, indeed, difficult to understand. But a curious case of partial agraphia has been recorded by Pitres.† In a syphilitic man, right hemiplegia without anæsthesia came on suddenly; it disappeared under treatment, leaving some rigidity, and there remained also right hemianopia with inability to write. Speech was never affected. The patient could copy in fac-simile, but could not write a single word, spontaneously or from dictation, with his right hand. Nevertheless he could write easily with his left, and then copy what he had written with his right hand. He could read either printed or written words with perfect facility. This strange case seems to prove the possibility of an isolated partial agraphia. A lesion separating the left arm-centre from the motor speech-centre, leaving the callosal fibres intact, affords perhaps the best explanation of the symptoms, but this or any explanation must be purely speculative; and it may be added, the possibility, and even probability, of more than one lesion prevents any conclusion from the combination of symptoms.

Persistent aphasia, motor or sensory, may be produced by any kind of lesion (even a tumour or the rare chronic senile softening), but it is due to acute softening more frequently than to hæmorrhage, on account of the greater frequency with which the former lesion involves the cortex; it is, however, sometimes due to hæmorrhage, and transient aphasia is often caused by this process in the central ganglia.

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* In the case recorded by Bernheim ('Revue de Méd.,' 1885, p. 625) of left hemianopia and mind-blindness in a left-handed man, alexia was necessarily present.

† 'Revue de Méd.,' 1884, p. 825.

The region of the cortex in which the speech-centres are situated is supplied by the middle cerebral artery (see Fig. 48, p. 64), and obstruction of this is the most frequent cause of aphasia. Motor aphasia is produced by the softening due to obstruction of the first branch of the artery, sensory aphasia by that of the fourth branch. Since the second branch supplies the chief part of the central convolutions, hemiplegia is often associated with motor aphasia. Another occasional association is visual loss. The occurrence of hemianopia with word-blindness has been already mentioned. Hemianopia is also sometimes associated with motor aphasia, but the affection of sight in such cases usually depends on a lesion near the internal capsule, generally a hæmorrhage or softening extending back as far as the posterior part of the capsule, so as to involve the fibres of the optic radiation. Softening in this position, *e.g.* near the thalamus, may also extend outwards to the first temporal convolution, or forwards and outwards, through the lenticular nucleus, to the island of Reil and third frontal convolution.

Loss of speech occurs in children from the same causes as in adults.* Some of these are exceedingly rare in childhood, while other causes are more common in early than in later life. One of the latter is tubercular disease, which is a comparatively frequent cause of aphasia in children, tubercular meningitis, probably in consequence of irritative inhibition (which may also cause motor paralysis), and, less commonly, tubercular growths. If recovery occurs from an organic lesion that has destroyed the motor speech-centre, the power of speech seldom remains absent for more than a few weeks, compensation by the right hemisphere occurring with great readiness. In cases of persistent aphasia in children it will generally be found, on careful investigation, that the defect of speech is part of a general mental defect, and not true, pure aphasia.

Aphasia may be due to functional disturbance of the brain as well as to organic disease. It may thus form part of various functional disorders, combined with other symptoms, and it may also occur alone. When right-sided convulsions occur from any cause, but especially from organic disease, there is often transient inability to speak, and this may continue for a quarter or half an hour, or even longer, after the fit. It is especially common when the convulsion begins in the face or tongue, and may occur when the spasm is very slight, and even when there is no convulsion, but merely a sensory discharge, tingling, &c. Aphasia is also met with as part of attacks of migraine, chiefly in those in which sensory disturbance occurs in the limbs of the right side. A patient who has such sensory disturbance, sometimes on one side, sometimes on the other, will have aphasia when the disturbance is on the right side and not when it is on the left. As an isolated symptom, functional aphasia occurs chiefly in children. It has been excited by emotion, by some peripheral irritation in the intestinal tract or elsewhere, and in the course of acute specific diseases, especially of typhoid fever. Many examples of this have been recorded. Transient aphasia was due, in one case, to the irritation of a tapeworm.* In cases of peripheral injury it is uncertain whether the chief influence has been reflex or emotional. A child became speechless during the division of a tendon without anæsthesia, and continued speechless for eight days.† Aphasia coming on during the course of typhoid fever has certainly been sometimes due to organic disease, for it has been associated with characteristic symptoms such as hemiplegia. But in other cases it has occurred alone, and after lasting for a few days, or for one or two weeks, it has passed away suddenly. Such cases suggest that the loss was due to an influence of the blood-state on the brain. It has come on as early as the first week, and, in one recorded case, it continued until the seventh week. It occurs chiefly in severe cases, but does not seem to render the prognosis more grave. The aphasia has generally presented the characters of the motor variety; very rarely those of the sensory form.‡ The variety of functional disturbance termed "dyslexia" has been already described.

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* Ninety cases of aphasia in children have been collected by Steffen ('Jahrb. für Kinderheilk.,' Bd. xxiii, p. 127).

Cases that can be regarded as congenital aphasia are extremely rare. In most instances there have been signs of a bilateral lesion of the brain, involving the motor centres for the tongue and lips, movement of which is usually distinctly defective. The cause is probably meningeal hæmorrhage during birth, symmetrical and seated over the lower part of the motor regions.

A very important question in connection with aphasia is its influence on testamentary capacity. It must depend, in any given case, on the ability to understand perfectly what is put before the testator, and to express at least assent and dissent with certainty. Word-deafness is incompatible with will-making, because it is impossible to know whether the testator really understands what is said to him. With perfect comprehension of spoken words, a valid will might be made if there was the power to express assent or dissent by signs, even if the testator was speechless.

The treatment of aphasia, so far as it is possible, is described in the chapter on softening of the brain.

GENERAL SYMPTOMS.

Besides the symptoms that depend on a derangement of the mental, motor, and sensory functions of the brain, cerebral disease may cause symptoms that are general, or consist in disturbance of other organs of the body. These may be conveniently considered together, although they bear different relations to the cerebral disease, and the relation of the same symptom may vary in different cases.

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* J. A. y Tusset, 'Estud. Clin.,' 1884, and 'Cent. f. Nervenh.,' 1884, p. 491.

† Demme, 'Wien. Med. Blatt,' 1884, No. 51.

‡ The subject of aphasia in typhoid fever has been discussed by Kühn, 'Deut. Arch. f. kl. Med.,' xxxiv, 56, and by Longuet, 'L'Union Méd.,' 1884, No. 60.

Temperature.—Elevation of temperature is frequent in cerebral disease, and is an instance of the difference of relation just mentioned. (1) It may be merely associated; the two symptoms, the cerebral disease and the pyrexia, being the result of a common cause. In ulcerative endocarditis we may have elevation of temperature from the alteration in the blood, &c., and cerebral symptoms in consequence of embolism in the arteries of the brain. In many cases we may be in doubt to what extent the pyrexia is due to the general or the cerebral disease, as, for instance, in general tuberculosis with a tubercular tumour of the brain or tubercular meningitis; and in pyæmia with secondary abscess of the brain or secondary meningitis. Recent observations, indeed, make it probable that the cause of the brain disease, *e. g.* meningitis (especially in the sporadic cerebro-spinal form allied to pneumonia and due to a blood-state), is the cause of pyrexia, formerly ascribed to the inflammation, much more frequently and to a much greater degree. But it is often most difficult to determine the extent to which this cause is the effective one, and it is equally important to bear in mind its possible operation. (2) The elevation of temperature may be due directly to the pathological process, which would have a similar effect if it were in any other situation. Meningitis is an example; the attendant pyrexia is generally the result of the inflammation. There are two important facts to be remembered regarding this consecutive elevation of temperature. First, it may be absent; the effect of the disease of the nervous system is to prevent the pyrexia which the morbid process would otherwise cause. Secondly, and connected with the last, is the fact that the pulse may not present the acceleration that usually accompanies pyrexia, and may even be abnormally infrequent. Hence the absence of pyrexia does not exclude intra-cranial inflammation, while elevation of temperature has additional significance if the pulse is not quickened. (3) The pyrexia may be the direct result of the cerebral lesion, that is of the disturbed function of the nervous system, not of the pathological process by which that function is disturbed. This effect is produced especially by lesions of the pons and medulla;[*] in these the temperature may rise to 104°, 106°, or 108°, and with the hyperpyrexia there is usually an abundant secretion of sweat. In some severe cases of hæmorrhage into other parts of the brain there is also an initial and considerable rise of temperature, to 103° and 104° (Bourneville). Such cases are usually rapidly fatal.

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[*] Experiments on animals (Ott, 'Journ. Nerv. and Ment. Diseases,' 1887, p. 152; Hale White, 'Brit. Med. Journ.,' 1889, i, 1401) show that lesions in the region of the corpus striatum cause a great rise of temperature, and that there are probably controlling centres in the cortex.

In some cases of meningitis the temperature becomes very high just before death, apparently from a similar mechanism. The pyrexia that accompanies the peculiar apoplectiform attacks in general paralysis of the insane is probably of this nature, since it is too brief, in proportion to its degree, to be ascribed to the pathological process causing the attack. Perhaps the brief fever that may attend the migrainous headaches of children is due to a similar mechànism. (4) The temperature may be raised as a secondary effect of the disturbance of cerebral function. When severe convulsions are frequently repeated, whatever be their cause, the temperature rises, apparently in consequence of the muscular exertion.

Depression of temperature is less common, and is met with chiefly at the onset of cerebral hæmorrhage. It is then accompanied by other signs of severe shock. When there is a large hæmorrhage, or when several hæmorrhages occur in quick succession, this fall may progress until death occurs, and the rectal temperature may fall to 90°. A moderate depression is sometimes observed at the onset, or in the course, of meningitis.

These effects may be variously combined, and the temperature in cerebral disease may consequently present very irregular and mysterious variations, now high and now low, without any corresponding variation in other symptoms.

Vaso-motor and trophic disturbance occurs most frequently in cases of hemiplegia, in the account of which it has been already described. It is also met with in meningitis and some other diseases, and may even be so pronounced as to be of diagnostic importance in chronic or subacute cases, the symptoms of which are otherwise equivocal.

The *pulse* may be increased in frequency when there is febrile disturbance, whether this is a consequence of the brain disease or merely associated with it; but, as we have seen, the acceleration that usually attends inflammatory pyrexia may be absent, and the pulse may even be less frequent than normal, although there is intra-cranial inflammation. As a direct effect of the brain disease on the cardiac centre of the vagus, the action of the heart may also be accelerated, retarded, or rendered irregular. This influence is exerted most frequently when the disease is near the medulla oblongata, but sometimes when it is distant. When intra-cranial disease raises the temperature to an extreme degree the pulse often becomes very frequent and soft. Irregularity is sometimes an early indication of meningitis, especially in children. Retardation to fifty, forty, or thirty beats per minute may occur in meningitis and apoplexy, and sometimes in cases of tumour, abscess, and of increased intra-cranial pressure, and also in some rare and mysterious functional affections. It may also be retarded in some toxic blood-states, as during influenza. The retardation is probably due to over-action of the centre that normally restrains the heart's action.

Respiration also is occasionally disturbed. When the cerebral functions are lowered, as in stupor or coma, the respirations are lessened in frequency and increased in depth. In meningitis and apoplexy (especially in cerebral hæmorrhage) the peculiar "Cheyne-Stokes breathing" sometimes occurs, and is usually the precursor of death. The respirations present a cycle of increasing and decreasing depth; the fall goes on to actual cessation of breathing, and after a pause slight respiratory movements recommence, at first scarcely perceptible, gradually increasing in intensity, until deep dyspnœal breathing marks the acme, and the respirations again lessen. During the pause the pupils often contract, and the heart's action becomes less frequent. The period of arrest lasts from five to forty seconds, and the duration of each cycle may be from fifteen to seventy-five seconds, and may vary from day to day. The number of respirations in each cycle never exceeds thirty. This peculiar phenomenon is met with in other diseases besides those of the brain. It occurs in heart disease (in connection with which it was first studied) and in uræmia, and has also been observed (chiefly in children) in rare cases of typhoid fever, scarlet fever, diphtheria, influenza, pneumonia, whooping-cough with inanition, and opium poisoning. In general maladies it is a less grave sign than in disease of the brain, and several cases of recovery are on record. In brain disease the patient is almost always comatose, but in other cases consciousness has been retained, and the symptom has been known to continue for as long as three months.[*]

A form of respiration which must be distinguished from that just described consists of periods of deep and energetic breathing, which begin suddenly; the respirations gradually lessen in depth until they cease, and after a period of stillness energetically recommence. The sudden commencement is the distinction of this form. In another variety, seen by Biot[†] in meningitis, the periods of deep breathing not only commence suddenly but end suddenly.

Many attempts have been made to explain the Cheyne-Stokes breathing, none of which are quite satisfactory. Walshe[‡] first suggested that it is due to lessened excitability of the respiratory centre, a theory afterwards stated, in more definite form, by Traube[§] and by Rosenbach.[||] It is difficult to understand how the gradual character of both the onset and the decline of the breathing can be due to simple lowered excitability, although, since rhythmical action seems a character of the centre itself,* it is just conceivable that the larger rhythm with gradual change may be due solely to the functional characteristics of the centre, working under altered conditions. The chief attempt to give a more complete explanation has been made by Filehne,† who found that the breathing could be produced in animals under the influence of morphia by making them inhale ether or chloroform. He observed, during the pause, an increase of blood-pressure with some pallor of the mucous membranes, and ascribed these to arterial spasm, due to stimulation of the vaso-motor centres by the asphyxial blood. He assumed that a lowered irritability of the respiratory centre renders it less excitable than the vaso-motor centre, that the consequent arterial spasm in the arteries of the medulla prolongs the stimulation of the respiratory centre (and also that of the vaso-motor centre) by hindering the access of the oxygenated blood. Thus the respiratory movements continue energetic after the blood has become well oxygenated. In support of this vascular theory he adduced two other facts. He was able to produce perfect Cheyne-Stokes breathing (even to the changes in the pupils) by gradually constricting and then releasing the arteries conveying blood to the head; and secondly, he found that, in a patient with this breathing, nitrite of amyl, which dilates the vessels, arrested the phenomena. Filehne's theory has been criticised adversely, and some of his facts have even been called in question, but chiefly by those who have vested interests in other hypotheses. On the whole, it may be said that, unless the simple rhythmical tendency of the depressed centre is adequate to produce the phenomena, they can be best explained by the assumption that this rhythmical tendency is modified by some other periodical influence, of which vaso-motor spasm is the only one which, according to our present knowledge, can be conceived as acting and adequate.‡ The gradual onset of the respirations may be due to the fact that the vaso-motor dilatation exceeds the normal (as it often does after contraction), and thus the quantity of blood reaching the respiratory centre lessens the stimulating influence of its quality.

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[*] West, 'Trans. Clin. Soc.,' 1890. In a correspondence which ensued in consequence of the publication of this case, a remarkable instance was mentioned ('Lancet,' 1890, i, 776) in which the phenomenon had persisted during several years.

[†] 'Sur la Resp. de Cheyne-Stokes,' Paris, 1878.

[‡] 'Diseases of the Heart,' 3rd ed., 1862, p. 345.

[§] Fraentzel, Report of Clinique, 'Berl. kl. Wochenschr.,' No. 27, 1869.

[||] 'Zeitschrift f. klin. Med.,' Bd. i, Heft 3. Rosenbach's theory is often spoken of as distinguishable from Traube's, from which it differs, however, only by ascribing the lessened excitability to altered "interchange of material in the cells."

Respiration is arrested at once by a sudden lesion in the respiratory centre of the medulla, and more slowly by disease that gradually impairs the action of the centre, such as the effusion of blood into the fourth ventricle. Indeed, the extensive connection of this centre with the brain, seen, for instance, in the readiness with which its action is altered by emotion, makes it sensitive to any considerable depression of the cerebral functions, and in many brain diseases death is thus brought about. Lesions near the centre may cause other disturbances of breathing than those described, such as simple irregularity, or paroxysmal cough, which may be semi-convulsive in character. On account of the gravity of lesions in this part, such disturbance of breathing is generally a fatal omen.

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* See 'Foster's Physiology,' 5th ed., pt. ii, p. 596.

† 'Berl. kl. Wochenschrift,' 1874, Nos. 13, 14, 32, and 35.

‡ The superficial analogy of the phenomena to the effects of "interference" in the action of two series of rhythmical events, as the waves of sound, can scarcely fail to occur to an observer, but of course no direct inference can be drawn from it.

In all conditions in which the cerebral functions are profoundly depressed, such as coma, secretion accumulates in the air passages; bronchial and tracheal râles are thus produced, and are often erroneously regarded as the indication of bronchitis. The accumulation is due chiefly to the depression of the respiratory centre, in consequence of which the secretion is not removed as in health, and its amount is perhaps increased by passive congestion. Such congestion of the lungs is very common in these cases, and is a frequent cause of death.

Digestive Organs.—Anorexia is common in febrile states, and in various organic diseases, acute or chronic, but no special significance can be attached to it. It is said that bulimia is sometimes met with, but it very rarely occurs as a symptom of organic lesions, although met with in hysteria and epilepsy.

Vomiting, however, is one of the most frequent and most important symptoms of intra-cranial disease. It is frequent in meningitis, tumour, abscess, at the onset of hæmorrhage, in cases of increased intra-cranial pressure, in various toxæmic states acting on the medulla, and also in the functional disturbance of hysteria. It may result from disease in any part of the brain, but is especially common when the cerebellum is affected. This symptom often occurs early, and may be produced by disease of very limited extent. The immediate cause of vomiting is doubtless an increased irritability of the gastric part of the vagus centre, and its occurrence in disease in various parts of the brain is another indication of the wide extent of the central relations of the vagus. It cannot be ascribed in all cases either to irritation of the membranes or to increased intra-cranial pressure, although these conditions are certainly capable of producing it.

In cerebral vomiting, food is generally rejected soon after it has been taken; there are no local gastric symptoms; no discomfort or pain is produced by the presence of food in the stomach, a symptom which almost always attends the quick rejection of food from gastric disease. Cerebral vomiting may be, and often is unattended by nausea, but this is not an invariable characteristic. In some cases nausea is distressing. Occasionally the patient vomits when there is no food in the stomach, but this is not common. The disease increases the irritability of the centre, but stimulation of the peripheral nerves by food is usually necessary to excite the act. The cerebral conditions that cause vomiting usually also cause headache, and the co-existence of persistent pain in the head with frequent vomiting should always raise a suspicion of cerebral disease. Optic neuritis is also a common result of the same cerebral condition, and the eyes should be examined in every suspicious case. For a long time there may be no other symptoms. It is most important, however, to note that in the early stage of disease, when the excitability of the centre is but little augmented, some exciting cause may produce the first vomiting, and the cause may seem adequate to account for it. Before the increased irritability of the centre reaches such a degree as to cause spontaneous vomiting it may be sufficient to lead to the rejection of an injudicious meal. Such explicable vomiting is often the first sign of intra-cranial disease, especially in children. The first symptom of a glioma of the pons was vomiting after swallowing a piece of slate pencil Patients who vomit without other indications of gastro-intestinal disturbance should be carefully watched. (See "Tumour.")

Constipation frequently attends intra-cranial disease, such as tumour, meningitis, &c., although it rarely exists until other symptoms are apparent.

Urinary Organs.—The urine is seldom altered in composition by organic brain disease, although polyuria rarely, and glycosuria occasionally, have been produced by disease of the pons or medulla oblongata. Albuminuria occasionally occurs from the same cause, and may be a transient effect of lesions elsewhere in the brain, or of meningitis. But albuminuria, it must be remembered, is a frequent concomitant of vascular lesions in the brain, because kidney disease is one of the most potent causes of vascular degeneration. The amount of albumen excreted in these cases is often greatly increased for a few days by a cerebral lesion.

Retention of urine and overflow incontinence are common in conditions of lowered cerebral function, from whatever cause; but there is not the paralytic incontinence that is met with in spinal disease (see vol. i, p. 225). Urine and fæces are often passed into the bed, &c., in cases of mental impairment, without any actual loss of power over the sphincters, and, as already stated, this symptom is always indicative of a considerable degree of mental change.

OPHTHALMOSCOPIC SYMPTOMS.

Morbid appearances in the fundus oculi are frequent in cases of organic brain disease, and are of great practical importance. Some of these are *associated*, the result of the cause of the cerebral lesion; others are *consecutive*, and are the effect of the brain disease. The two kinds are sometimes conjoined.

1. The *associated* changes are due to the constitutional condition, that is, to the ultimate cause of the brain disease, and are sometimes of the same nature as the cerebral lesion. The following are the most important:—(1) Albuminuric retinitis, acute and chronic; it is associated chiefly with arterial degeneration, causing either cerebral hæmorrhage or softening from thrombosis. (2) Syphilitic disease, usually choroiditis, or the atrophy that such inflammation leaves.

This is often to be seen in cases of brain disease due to either inherited or acquired syphilis. (3) Tubercular disease. Tubercles of the choroid are sometimes met with in cases of tubercular meningitis. Very rarely massive tubercle forms within the eye, in cases of tubercular growths in the brain. Other forms of associated growth are so rare as not to be of practical importance. (4) Vascular lesions in the retina sometimes coincide with similar lesions in the brain. Thus embolism of the central artery may occur in the same patient, and even at the same time,* as embolism of a cerebral artery. Miliary aneurisms on the retinal arteries have been observed to coincide with cerebral hæmorrhage, probably due to similar aneurisms in the brain. They are, however, extremely rare.† Hæmorrhages in the retina are more common, and are of some significance. They occur especially in albuminuria, in gout that has profoundly affected the system, in leucocythæmia, profound anæmia, purpura, and also in ulcerative endocarditis, and other forms of septicæmia. In the latter they often have white spots in the centre, and are the result of capillary septic embolism. Hæmorrhages, identical in aspect, are sometimes found in the same cases in the pia mater. Another associated lesion (to which we shall return) is simple atrophy of the disc, which sometimes coincides with degenerative lesions of the brain.

2. The *consecutive* changes, which are not merely associated with the cerebral disease, but are its consequence, comprehend neuritis and atrophy.

Neuritis or "papillitis," inflammation of the intra-ocular end of the optic nerve, is manifested by swelling and increased vascularity of the "optic papilla." It will be remembered that the end of the nerve is visible in the fundus as the "optic disc," the boundary of which is the oval opening in the sclerotic with the corresponding opening in the choroid. The fibres radiate from the nerve on to the retina, but not equally on all sides, being few on the temporal side. The nerve-elements are so translucent that they do not obscure the edge of the disc, except sometimes, to a slight extent, above and below, where they are more numerous. The disc is rarely uniform in its surface, because the separation of the nerve-fibres leaves a central hollow, in which there are few or no vessels, and which is therefore pale, while the periphery of the disc has a rosy tint from the minute vessels that lie among the nerve-fibres (Fig. 69). This central "physiological cup" varies much in size, and may be absent. The vascular portion of the disc also varies in extent (inversely as the cup), and varies so much in tint that no inference can be drawn from its colour unless this is observed to change in the course of time. Mere congestion, therefore, consisting only of increased vascularity of the disc, is very difficult to recognise, and although it probably occurs, it is rare, as an isolated condition, and is of small practical importance. An actual pathological change, however slight in degree, is usually attended by some swelling of the papilla, and especially by lessened transparency of its structures. The effect of this change is first to lessen the sharpness of the edge of the disc, and then to obscure the edge altogether (Fig. 69). It is to this point, therefore, that attention must be chiefly directed. The effect of a morbid change on the distinctness of the edge is greater when the disc is examined by the direct than by the indirect method; on the other hand, if the indistinctness is apparent and not real—is due to the tint of the disc being nearly that of the choroid, and not to lessened transparency of the structures in front of it—the edge appears more distinct in the direct than in the indirect method of examination. This is a very important practical point. In the early stage of neuritis the edge of the disc, seen by the indirect method, may appear a little blurred, and surrounded by a pale halo, while by the direct method the halo is resolved into a striated semi-opaque layer completely concealing the edge. The early change is greatest on the nasal side, which may be obscured when the temporal edge of the disc, on which there are few nerve-fibres, is still sharp. As the change advances, it involves the whole circumference of the disc, and the swelling rapidly encroaches on the physiological cup, which is ultimately obliterated (Fig. 69), although a slight depression usually remains in the middle of the swollen papilla. The prominence of the swelling is readily recognised by the apparent

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* See 'Med. Ophthalmoscopy,' Case 47 (p. 332, 2nd ed.).

† For an instance, see 'Medical Ophthalmoscopy,' pl. xii, fig. 1, and Case 42 (2nd ed., p. 326).

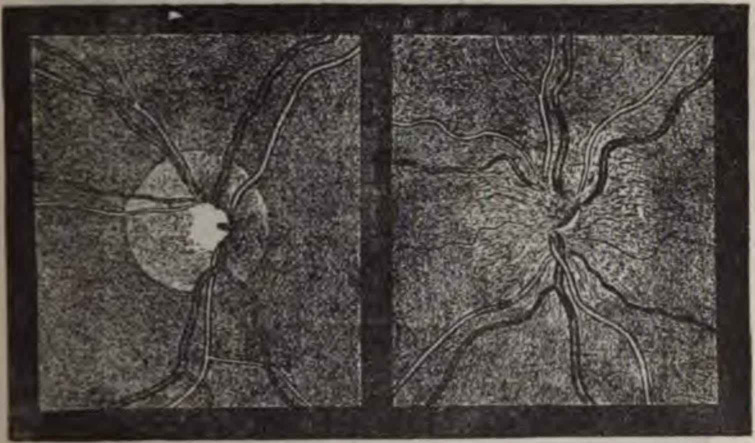

FIG. 69.—Commencing optic neuritis, from a case of caries of the sphenoid bone with secondary meningitis. The left figure shows the normal right optic disc, with clear outline, and a deep central cup. The right figure, of the left papilla, shows well-marked neuritis; the edge of the disc is concealed by a swelling which extends beyond the normal limits of the disc. The central cup is encroached on, but not yet quite obliterated; before the neuritis commenced the cup was similar to that in the other eye. Some of the vessels are partly concealed at their emergence, and the veins lose their central reflection as they curve down the sides of the swelling.

change in relative position of the structures that are on different levels when the observer moves his head from side to side or up and down in the direct examination, or moves the lens in a like manner in the indirect examination. The prominence is also shown by the loss of the central reflection of the vessels where they course down the sides of the swelling, and their plane ceases to be at right angles to the line of the observer's vision. The tint of the swollen papilla becomes a full red, or, more commonly, a greyish red, to the indirect examination, but the direct method shows a fine striated vascularity. As the prominence increases, the swelling becomes wider in extent, until it may be two or three times the diameter of the normal disc. White spots may appear on its surface (Fig. 70), due to the accumulation of products of degeneration. At first the retinal vessels present little change in size, but as the swelling increases, the compression causes the veins to become broader and the arteries narrow, and extravasations of blood may be visible on the surface or margins of the swollen

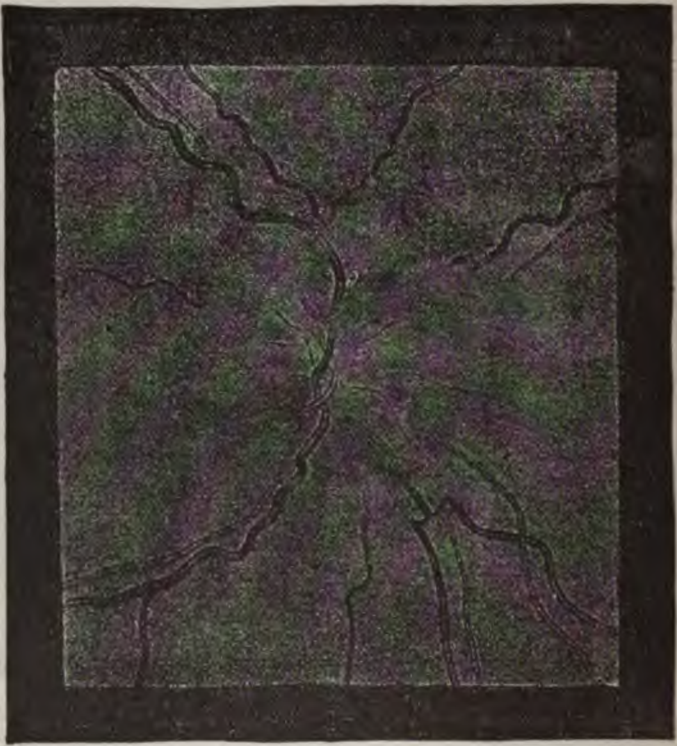

FIG. 70.—Optic neuritis in cerebral tumour. The swelling is great, and occupies an area at least twice the diameter of the normal disc. The arteries are concealed in the substance of the new tissue, and the veins are tortuous, and lose their reflection as they pass down the side of the swelling. The products of degeneration of the nerve-fibres have caused white spots near the margin of the inflamed papilla.

area. The process varies much in the rapidity with which it is developed; it may reach a considerable intensity in a fortnight, or be still moderate in degree at the end of three or four months. As a rule, the more quickly it is developed the more intense does it become. Commencing subsidence is indicated by a diminution in the vascularity, still greater contraction of the arteries, and later, contraction of the veins also. If the degree of neuritis has been considerable, the swelling remains, for some time, pale and soft-edged, and slowly sinks until the edges of the disc appear. For a long time the substance of the disc is occupied by new tissue, which, with the narrowed arteries, affords evidence of the preceding inflammation. Other indications of this are often to be seen in damage to the adjacent edge of the choroid. Only when the neuritis has been moderate in degree does the disc resume a perfectly normal aspect. When much new tissue has been formed, this, by its cicatricial changes, leaves the disc white and atrophied, in the condition of "consecutive atrophy," or "neuritic (papillitic) atrophy," as it is also termed. When the papillitis has been very intense, and the swelling wide in area, the adjacent retina may suffer in its nutrition, and products of degeneration may remain as white spots, especially near the macula lutea, simulating the aspect of albuminuric retinitis.

The substance of the swollen papilla presents, under the microscope, distended vessels, numerous capillaries and nuclei, with accumulations of leucocytes in the tissue and perivascular sheaths. In the early stage, the nerve-fibres and connective-tissue elements may be separated by œdematous effusion. Subsequently the nerve-fibres present degenerative changes, moniliform enlargements containing myelin globules, and aggregations of the products of degeneration into "granule-corpuscles," while the walls of the arteries may be conspicuously thickened. Similar, although slighter, changes may be traced back in the substance of the optic nerves, or in their sheaths, to the chiasma, and they may be even more intense in front of the chiasma than they are midway between this and the eye. The outer sheath of the optic nerve is often distended by liquid, so as to form a pyriform swelling behind the globe. The space within this sheath is continuous, behind, with the subarachnoid space around the brain, and in front, with the lymphatic spaces in the optic papilla.

A slight and even considerable degree of optic neuritis may cause no symptoms. Acuity of vision may be unimpaired, colour vision normal, the visual fields unrestricted. But when intense, sight suffers in each of these characters, and may be entirely lost. The damage to vision is often greater during the stage of subsidence than it is during the active period of inflammation, probably because the nerve-elements suffer from the cicatricial contraction of the tissue-elements formed during the inflammation. But the affection of sight is not necessarily due to the visible papillitis; it may be the result of more intense inflammation behind the eye, or the effect of the intra-cranial disease. To decide this, the degree of neuritis and the degree of affection of vision must be compared, and the character of the failure of sight must be considered. The latter is described in the chapter on affections of the optic nerve. After the neuritis has quite subsided some improvement in sight often takes place, from the recovery of some of the damaged fibres. But there may remain considerable amblyopia, and even absolute blindness, and often very irregular changes in the fields of vision, both for white and coloured light.

Optic neuritis may result from many diseases of the brain, but its most frequent cause is tumour, and a considerable degree of neuritis is rarely due to any other cause. Next in frequency is meningitis, especially affecting the base of the brain, but the papillitis caused by meningitis is usually less intense than that of tumour, perhaps on account of the briefer duration of the disease. Cerebral abscess is another cause. Optic neuritis scarcely ever results from cerebral hæmorrhage or from thrombotic softening, but it has been several times observed in cases of softening from embolism when the source of the obstructing plug was acute endocarditis, perhaps because the material carried from the valves is of an irritant septic character, and determines a greater degree of secondary inflammation in the softening produced. It has also been met with in a case in which no naked-eye lesion of the brain existed, but in which the microscope revealed general slight inflammatory changes—diffuse cerebritis. In all these diseases the neuritis is, as a rule, double, although it may develop more rapidly in one eye than in the other. Occasionally it is one-sided, and then it is generally on the side opposite the lesion. Unilateral optic neuritis is much more frequently due to various diseases at the back of the orbit, or at the optic foramen, than to disease of the brain itself.

It must be remembered, however, that optic neuritis may result from other causes than intra-cranial disease. It is very seldom primary; but it occurs in chlorosis, albuminuria, lead-poisoning, anæmia, and after certain acute fevers, especially scarlet fever and typhoid; in many of these conditions its occurrence is associated with some cerebral symptoms. When neuritis preponderates over the other changes in albuminuria, for instance, there is usually much pain in the head, and in lead-poisoning it is usually associated with the acute cerebral symptoms which have been called "encephalopathia saturnina." This fact sometimes increases the difficulty in diagnosis.

The mechanism by which optic neuritis is produced is a subject on which various opinions have been held.* It is sufficient here to say that the early theory that neuritis is due to increased intra-cranial pressure, acting mechanically, has proved erroneous. I have known slow increase of intra-cranial pressure to separate the sutures without causing papillitis. In a large number of cases there is distinct evidence of a descending inflammation, either along the trunk of the optic nerve or along its sheath, and in cases of meningitis such descending inflammation is invariable. The distension of the optic sheath is not essential for the production of neuritis; it is sometimes absent, and its occurrence is related especially to the presence of an excess of subarachnoid fluid. The signs of mechanical "strangulation," which are to be observed in cases of intense neuritis, are no evidence that the inflammation was caused by any mechanical process. The cause of the strangulation is the compression of the veins by the inflammatory products within the swollen papilla, and not, as was at first thought, compression within the sclerotic ring or behind it, by the distension of the sheath. Within the ring, the vessels after death never exhibit any sign of narrowing, whereas within the papilla their compression is usually distinct. The conclusion is that optic neuritis is probably rarely due to a single factor, that the most potent element is the descent of a process of tissue-irritation, which, when it reaches the papilla, sets up a more intense inflammation; that in some cases this factor is alone effective; that in many others its influence is aided by the effect of distension of the sheath, hindering the escape of effete liquids, increasing the œdema, or even conveying irritating material. The distinction between optic neuritis and "choked disc" is one of degree, not of cause. So far as optic neuritis has any single significance, it is that of the presence of an irritative process within the skull.

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* A full account of these will be found in 'Medical Ophthalmoscopy,' 3rd ed.

There is an important relation between the chronicity of the neuritis and that of the intra-cranial process. A chronic cerebral process may cause an acute neuritis, but a chronic neuritis never results from an acute process, and the degree of the chronicity of the neuritis is an indication of the degree of chronicity of the intra-cranial disease. When the cerebral process begins to improve, the neuritis lessens, and the commencing subsidence of the neuritis is often the first indication of the improvement in the brain lesion. Hence the process often gives valuable prognostic information as well as diagnostic guidance.

Atrophy.—Of the three forms of atrophy of the optic nerve, *primary*, *secondary* (to disease of the nerve further back), and *consecutive* (to neuritis), only the latter two occur as a result of uncomplicated intra-cranial disease. Apparent exceptions to this are disseminated sclerosis and general paralysis of the insane: in both of these affections primary atrophy may occur, but in these there is wide-spread degeneration, usually involving the spinal cord as well as the brain, and the atrophy must be regarded as associated with, rather than the result of, the brain disease. Consecutive (neuritic or papillitic) atrophy, the characters of which have been already described, has the same causes and the same significance as the inflammation of which it is the consequence.

Secondary atrophy is the result of damage to the optic nerve behind the eye, or to the optic chiasma. Its characteristic is that the signs of atrophy follow instead of accompanying the failure of sight. Especially important, on account of the diagnostic difficulty it presents, is the form of secondary atrophy that follows temporal hemianopia of slow onset, due to chronic disease at the optic chiasma. The causes of this form of atrophy will be described in the chapter on diseases of the optic nerve.

DISEASES OF THE CRANIAL NERVES

AND DERANGEMENT OF THEIR FUNCTIONS.

Many symptoms of organic brain disease consist in derangement of the functions of the cranial nerves, and it is convenient to consider these together with the diseases of the nerves themselves. Such an arrangement is not strictly logical, but its practical convenience is great, since much needless repetition is thereby avoided, and those symptoms can be described together that are of the same character, and have to be considered together in practical diagnosis. The paths of the cranial nerve-fibres within the brain have been already described, and need not be here repeated, except in so far as they have a bearing on special points.

OLFACTORY NERVE—AFFECTIONS OF SMELL.

The olfactory bulb contains many nerve-cells, and has been thought by some to be analogous to the nuclei of the other cranial nerves, by others to be a detached portion of the cerebral cortex. The bulb is connected with the cerebral hemispheres by the olfactory nerve, the central connections of which have been described at p. 56. We have seen that each nerve seems to be related to both hemispheres, since the sense of smell on one side may be deranged, not only by disease of the hemisphere (uncinate gyrus) on the same side, but also by a lesion of the hinder part of the internal capsule on the opposite side, and probably also by disease of the convexity.

The sense of smell includes more than is popularly assigned to it. It includes not only the recognition of odours, but also that of flavours. When the olfactory nerves are influenced through the anterior nares the sensation is termed an "odour;" when through the posterior nares it is termed a "flavour." In the latter case the sensations are blended with those of taste proper, bitter, sweet, sour, &c., and a mixed sensation is the result. If the sense of smell is lost, all perception of flavours is lost with it, but the true gustatory sensations remain. The two functions of the olfactory nerve seem to be separated at the cortex, and the impressions of flavour to be perceived through the same centre as those of taste, since this relation is always preserved in the aura of epilepsy. Hence patients who have lost smell usually say that they have also lost taste, and sometimes they only mention the loss of "taste," which is to most persons the more serious deprivation.*

In testing the sense of smell, care must be taken to employ only substances (such as musk, asafœtida, or oil of cloves) that act on the olfactory nerve alone, and it is best to use such odours as are readily identified and named. If volatile pungent substances are used, such as ammonia or acetic acid, which also stimulate the fifth nerve in the nasal mucous membrane, the patient may perceive that which he cannot smell.

Loss of the sense of smell, anosmia, is less frequently due to a lesion of the nerve than to disease of the mucous membrane of the nose, chronic inflammation, nasal polypus, &c. It may also be caused indirectly by paralysis of the fifth nerve and consequent trophic changes in the membrane, or dryness from lessened secretion. Excessive secretion may also produce it. If the nose be filled with rose water no smell is excited, and smell remains in abeyance for some time. The olfactory nerves may be damaged by falls or blows on the front or back of the head; the delicate filaments are torn from the bulb, or are lacerated in their passage through the ethmoid bone. The olfactory bulbs sometimes suffer, by compression or inflammation, in cases of tumour in the anterior fossa of the skull, or elsewhere within the cranial cavity, even in the cerebellum, caries of adjacent bone, and local meningitis, syphilitic or other. They may also be compressed by internal hydrocephalus.† In these cases the bulbs may be distinctly involved in the disease, or may be flattened and wasted. Tumours have been supposed to cause an olfactory neuritis, analogous to optic neuritis. In locomotor ataxy, anosmia occasionally occurs, although not frequently. Primary atrophy of the bulbs is occasionally met with in old age (Prévost). Lastly, excessive stimulation of the olfactory nerves has caused their paralysis, just as blindness has resulted from a very brilliant light; in several cases, after exposure to an exceedingly strong odour, the sense of smell has been permanently lost. Thus an officer in Ireland superintended the emptying of a cesspool at the bottom of which some weapons were said to be concealed; the odour was most offensive. Next day he found that his sense of smell was gone, and it never returned. Other similar cases are on record. It has been conjectured that the odour causes congestion and hæmorrhage into the mucous membrane, because, if the musk deer-hunters do not stuff up their nostrils before extracting the musk gland, they suffer from epistaxis.[*] But it is unlikely that capillary hæmorrhage into the mucous membrane would cause complete anosmia, or would occur without epistaxis. More probably the loss of smell is the direct result of the over-stimulation on the nutrition of the nerve-elements.

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* The word flavour originally meant smell. The nomenclature given above is that of Milton, who, speaking of wine, says,—

"The flavour or the smell,
Or taste that cheers the hearts of gods or men."
Samson Agonistes, 544.

Skeat, it is true, thinks Milton may have meant colour, "flavor" in low Latin meaning yellow; but no contemporary example of this use is given.

† Quincke, 'Cor. Bl. f. Schweiz. Aerzte,' 1882, No. 9.

In diseases of the cerebral hemisphere loss of smell is rare. It has occurred with symptoms of embolism of the middle cerebral (Hughlings Jackson), perhaps from damage to the root-fibres at the commencement of the fissure of Sylvius, since the loss of smell has been on the same side as the lesion. It has also been observed, on the opposite side, in cases of disease of the "sensory crossway," at the hind part of the internal capsule,[†] and in at least one case of extensive disease of the convexity.[‡] Functional loss occurs only in hysterical hemianæsthesia. The olfactory nerves are sometimes congenitally absent. Dr. Sharpey, in his lectures on physiology, used to mention the instance of a chief scavenger of the city of Leipzig, who was known to have no sense of smell, and in whose brain no trace of olfactory nerves could be found. A few similar anatomical facts are on record; in most cases a minute grey prominence marked the place from which the nerve normally springs. Congenital absence has been known to descend from father to son (Breschet). It does not seem certain, however, that the absence of the nerves invariably entails absence of the sense.[§] Possibly, as suggested by Duval,[||] the fifth nerve subserved the olfactory function.

Injury may cause partial loss, sometimes curious. One intelligent man, after concussion, could only perceive a certain "scent," and of flavours, that only of raspberry jam, even strawberry jam being unrecognised. The state was permanent.

The diagnosis of an affection of the nerve depends first on the detection of the loss of smell, and secondly on the exclusion of disease of the mucous membrane. In the latter case purulent discharge has generally occurred at some period, and a history of such discharge always affords strong reason for suspecting a local cause. Loss of smell, with ability to recognise flavours, is always due to such disease, sometimes partial in extent. The importance of a local examination is obvious. A lad was once sent to me who was said to have lost smell, taste, and hearing; I was asked where the brain disease was likely to be to cause these effects. There were scrofulous inflammation of the nasal mucous membrane, caries in both ears, and neither loss of taste proper nor any evidence of intra-cranial mischief.

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[*] Althaus, 'Lancet,' 1881, vol. i, p. 771, where some interesting information on the history of our knowledge of anosmia will be found.

[†] See note on p. 20.

[‡] The case of Demange, p. 18.

[§] Case of Bernard, quoted by Althaus, 'Lancet,' 1881, i, 772; and of Lebec, 'Prog. Méd.,' No. 48, 1883.

[||] 'Bull. de la Soc. de Biol., Paris,' No. 17, 1883.

Besides the ordinary tests of smell, the nerves can be stimulated by voltaic electricity, a phosphorus-like odour being perceived. This is proof of their integrity, but the application is very painful unless the nose is filled with water, or, better, with a weak saline solution, in which the rheophore is placed. In health the order of response has been thus found to be similar to that of other nerves: (1) KCC, (2) AOC. The induced current has no effect. An electrical examination can very seldom be necessary. The fact that disease of the fifth nerve may cause considerable anosmia must not be forgetten in diagnosis.

The prognosis of disease of the olfactory nerve is generally unfavorable. Considerable improvement has been observed only in cases of syphilitic disease of short duration, and in some traumatic cases.

The treatment is mainly that of the cause of the anosmia. The local application of strychnia to the mucous membrane has been recommended—as a solution in olive oil (Brunniche), or as snuff, $\frac{1}{24}$ grain mixed with sugar (Althaus). In cases in which there is reason to believe that the active disease has ceased, voltaic electricity has been recommended, the positive pole being placed behind the mastoid process, the negative to the nasal bones. Only a few cells can be used, or unpleasant giddiness will be produced. In one recorded case, after a fall on the back of the head, considerable improvement followed two applications (Jacob). Of course, all that electricity can do is to excite the nerve-elements that are structurally perfect, but for some reason functionally inactive, and in most cases of anosmia it is useless. Iodide of potassium has also been recommended, but its value, except in cases of syphilitic origin, is doubtful.

Increased sensitiveness of the olfactory nerves—"olfactory hyperæsthesia," "hyperosmia"—is occasionally met with. It occurs chiefly in hysteria, in which remarkable, almost animal, acuteness of the sense is sometimes present, so that not only objects but persons have been discriminated by this means. In insanity the same condition is sometimes met with. It is usually associated with, and has to be distinguished from, an altered appreciation of odours, shown in the abnormal enjoyment of, or disgust at, the odours which are recognised with natural or preternatural acuteness.

The condition rarely calls for special treatment. Morphia in snuff or hypodermic injection has been suggested, but the local application of cocain will probably be found more effective.

Subjective Sensations of Smell.—Subjective sensations of smell occur from central disease, or from irritation of the nerve. In the insane, olfactory hallucinations occur, though less commonly than those of the optic or auditory nerve. Schlager met with them in five cases out of six hundred. In epilepsy subjective sensations of smell occur as occasional warnings of fits, and disease in such a case has been found to involve the olfactory region in the anterior part of the temporo-sphenoidal lobe. It was so in a case of tumour, recorded by Sander, and also in one of softening of this region.[*] In a case recorded by Dr. Hughlings Jackson and Dr. Beevor[†] the aura consisted of a crude sensation of smell and the so-called "dreamy state." There was no impairment of the sense of smell, but the autopsy revealed a tumour involving the anterior part of the right temporo-sphenoidal lobe. A subjective sensation (usually resembling that of phosphorus, and such as is produced by electrical stimulation of the nerve) sometimes precedes loss of smell from organic disease, as by a tumour. It was so in a case of loss of smell in tabes recorded by Althaus. Doubtless the sensation is due to the irritation of the nerve-elements by the morbid process which ultimately destroys them.

Perversion of the Sense of Smell.—This is a rare condition, which occasionally results from irritation of the nerve or central organ. In a case recorded by Legg, some time after an injury to the head, all substances "tasted" of gas or paraffin, and there was marked diminution in the acuteness of the sense of smell. In a case of tabes recorded by Erben[‡] there was perversion of smell and a similar condition of taste.

OPTIC NERVE AND VISUAL SYMPTOMS.

The central relations of the optic nerve have been described at p. 54, and the cortical centres for vision at p. 21. The fibres from the region of the macula lutea are at first in the outer part of the nerve, and reach its axis near the optic foramen. Each optic tract contains the fibres from the same-named half of each retina, *i. e.* from the temporal half of the retina on the same side, and the nasal half of the retina on the other side, and hence disease of the tract causes loss of vision in the opposite half of each field, the temporal half of the one, the nasal half of the other. This is termed ***homonymous hemianopia***,[§] or *lateral hemianopia*.

Why do more fibres cross to the opposite tract than pass to the tract on the same side, if the two sets come from equal retinal areas? Because the prominence of the nose shuts off the rays from the peripheral part of the temporal half of the retina, and the power of sight extends but little further than the area habitually stimulated. Hence, while the retinal halves are of equal size, the functional area is smaller on the temporal side, and fewer nerve-fibres proceed from it.* The fibres from the temporal side are those that do not cross, and hence the crossing fibres are more numerous than those that pass in the tract without decussating. The fibres which cross occupy the middle of the chiasma, the direct fibres lie on each side. Fibres from the macula lutea and region around it pass, in most individuals at least, to both optic tracts. The division between the two halves of the retina (from which fibres pass to the respective tracts) is generally in the vertical line of the macula lutea, but it probably varies in its precise direction in different individuals, and seems sometimes to be slightly oblique. These two facts will be considered more fully in the description of the symptoms. The fibres which pass from one tract to the other by the posterior part of the commissure (and are probably connected, by the internal geniculate bodies, with the corpora quadrigemina) have not, at present, any medical importance.

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[*] Hamilton, 'New York Med. Journ.,' June, 1882.

[†] 'Brain,' vol. xii, 354.

[‡] 'Wien. Med. Blätter,' 1886, pp. 43, 44.

[§] It was formerly called "hemiopia," but the anomaly of the use of the term "half-sight" in the sense of "half-blindness" has led to a general endeavour to employ the more exact "hemianopia" or "hemianopsia." Although it is seldom desirable to change a familiar word, the alteration is trifling and removes an inaccuracy.

We have seen that the visual path passes, on each side, by the optic tract to the optic thalamus, and thence by the optic radiation to the cortex of the occipital lobe. This constitutes a half-vision centre which seems to be double, for light and for colours. The two half-vision centres are probably blended in a higher visual centre in front of the occipital lobe. The arrangement is, however, very complex; in each centre both fields are represented, but chiefly that of the opposite eye. Each higher visual centre can supplement its fellow to some extent and compensate its loss, but in the half-vision centres there is no power of supplemental action, and no compensation for loss is possible. The effects of destruction of the path or centre (when the results of the slighter damage have passed off) are permanent. This is strikingly shown by Fig. 80.

ETIOLOGY.—The numerous affections of the optic nerve within the eye do not come within the province of this book, nor does the primary atrophy, which so often accompanies certain degenerative diseases of the spinal cord, and occasionally complicates similar diseases of the brain. Optic neuritis, or "papillitis," such as results from intra-cranial tumours, is rather a general symptom of cerebral disease than a special affection of the nerve, and has been considered with other symptoms of the same class (p. 132). Moreover, many diseases of the nerve itself behind the eye, such as interstitial neuritis, or hæmorrhage into the substance of the nerve, also fall chiefly into the province of ophthalmology.

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* Semi-decussation of the optic nerve is not the invariable rule in the animal kingdom. In some creatures there is a total decussation. The amount of decussation seems to be roughly proportioned to the amount of separation of the fields of vision. In man the fields half overlap when the eyes are directed forwards, and there is a half-decussation. In those animals in which the fields are totally distinct and cannot be made to overlap, as fishes, there is a total decussation.

The diseases that are of medical significance affect the nerve, the chiasma, the tract, or the path from this to the cortex, or the cortex itself. This classification corresponds to an important difference in the symptoms produced.

(1) The *nerve* itself may be damaged by tumours within the orbit or within the skull. The intra-cranial course of the nerve is indeed so short that it is seldom thus affected, but it is occasionally compressed by a tumour of the pituitary body, or by one springing from the bone. The nerve may be damaged by an aneurism of the ophthalmic artery within the orbit, or of the internal carotid within the skull. It is occasionally the seat of inflammation, interstitial or arising in the sheath or spreading to it from an adjacent focus, such as caries of the sphenoid bone or an orbital abscess. It often becomes inflamed in meningitis, but symptoms result chiefly through the agency of papillitis. A rare but important form is rheumatic inflammation. Of this I have seen several instances, chiefly in women, and two had previously suffered from a neuritis of the facial nerve, and all from other manifestations of rheumatism. The symptoms followed exposure to cold. In some cases the nerves to the eyeball muscles were also involved, and from this it is probable that the seat of this inflammation is at the back of the orbit. The optic nerve is occasionally injured in falls on the head, either by laceration at the optic foramen or by hæmorrhage.

(2) The *chiasma* is damaged most frequently by tumours arising in its neighbourhood, especially by those springing from the pituitary body, sometimes by tubercular or syphilitic growths, in its substance or outside it, rarely by chronic inflammation in its vicinity, commencing in the dura mater and leading to thickening of the tissues about the commissure. Another occasional cause of damage is internal hydrocephalus; the distended infundibulum of the third ventricle presses on the middle of the chiasma and flattens it, as Cheselden noted a century and a half ago.* It is probable that it is sometimes the seat of interstitial inflammation, of which inherited gout may be the apparent cause. Similar symptoms occur in rare cases of tabetic atrophy of the optic nerve, and may develop slowly or rapidly. This process may conceivably be sometimes more intense at the chiasma than elsewhere, even in slow atrophy, and may even assume the character of a secondary inflammation in some cases (see "Tabes," vol. i). In one case which I have recorded the characteristic loss of sight came on quite suddenly, and has been observed in other cases of tabes; it suggests a secondary vascular lesion. Interstitial hæmorrhage has been actually observed.†

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* 'Phil. Trans.,' No. 337, p. 281.

† For details of lesions see Wilbrand, 'Hemianopsie,' Berlin, 1881, and Hill Griffith, 'Med. Chronicle,' January, 1887.

(3) The *optic tract* is chiefly damaged by tumours either of the base or springing from the inner part of the temporo-sphenoidal lobe, which may either compress the tract or invade it. An instance of such compression is shown in Fig. 71, although it is doubtful whether the hemianopia, from which the patient suffered, was thus produced, since the disease began in the hemisphere in the neighbourhood of the optic radiation, and the hemianopia was an early symptom. (The cerebral growth is figured in the chapter on "Intra-cranial Tumours.") If the growth *invades* the tract, it generally spreads also to the crus, as in a case in which such a tumour caused first hemianopia and then hemiplegia.* The tract is seldom damaged by softening or hæmorrhage; softening in the lenticular nucleus has invaded the tract, and partial softening has been observed to result from thrombosis of the internal carotid. It is sometimes the seat of an islet of disseminated sclerosis.

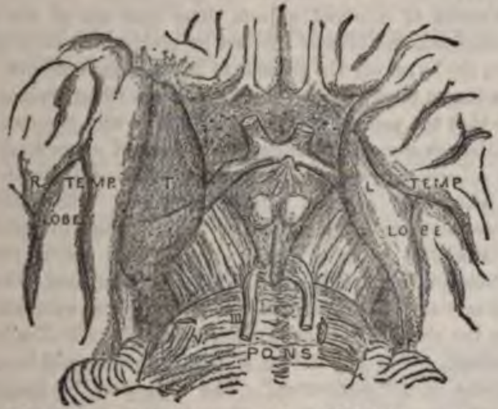

FIG. 71.—Tumour of right temporo-sphenoidal lobe, compressing the right optic tract, but previously interrupting the optic radiation. (See chapter on "Intra-cranial Tumours.")

The intra-cerebral path may be damaged by softening, hæmorrhage, tumours, or traumatic lesions. These may involve the thalamus, posterior part of the internal capsule, white substance of the occipital lobe, or cortex.

INVESTIGATION OF THE FUNCTIONS OF THE OPTIC NERVE.—The manifestations of disease consist in alterations in the fundus oculi, and in impairment of vision or of the reflex action of the iris to light. All these should be carefully examined in every case; and it is often necessary also to ascertain the state of refraction in the eye, lest a defect of sight should be ascribed to disease of the visual path or centre, when it depends solely on an error in refraction. This is of great importance, especially since a considerable deviation from the normal refraction is apt to set up other functional disturbances, pain,

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* 'Cent. f. med. Wiss.,' 1878, No. 31.

&c., which may lead to a suspicion of organic disease. The suspicion may be apparently confirmed by the impairment of sight. On the importance of an ophthalmoscopic examination it is needless to insist; without it, amblyopia may be ascribed to brain disease when it is of purely ocular origin, and many morbid processes in the brain reveal themselves in the optic papilla. The more important changes in the fundus have been already mentioned (p. 131).* The mode of testing the action of the pupil will be described in the chapter on "Diseases of the Third Nerve."

The *examination of vision* includes several points, each of which needs careful investigation. (1) The acuity of vision, for which test-types are employed, and the result is expressed in a fraction of which the denominator is the distance at which the type can be read by a normal eye, and the numerator is the farthest distance at which it can be read by the patient. (2) Colour vision may be tested in two ways: (*a*) by the method of "confusion," in which the patient selects from a series of coloured objects those that are of the same kind of colour as the standard, and reveals a defect by picking out tints that are essentially different; (*b*) by asking the patient to name certain colours. This is open to the fallacies that a name may be given accurately when a colour is not rightly perceived, and that the patient may not be familiar with the names of colours. The former fallacy is especially dangerous in cases of congenital colour-blindness, in which colours may appear different, and be associated with certain names in the mind, when they are not rightly perceived. The method of naming colours is, however, more often useful in medical than in ophthalmic work. If a patient is known to have previously had good colour vision, or if one eye only is affected, and the vision with it can be compared with that of the other eye, the method of naming colours often gives valuable information, especially as to the character of the sensation excited. (3) The fields of vision must be examined, and if any defect is found, they should be mapped out, if possible, by means of a "perimeter." The field may be roughly tested by moving some object midway between the observer and the patient, the eye tested being fixed on that of the observer, who can thus readily detect any movement of the patient's eye from the fixing-point towards the object, and at the same time can use his own field of vision as a standard. For a very rough examination the hand may be employed, but it is better to use a small piece of white paper one third of an inch square, fixed on the end of a dark-coloured rod, such as a penholder. With a little care very exact observations can thus be made, although the result cannot be recorded so precisely as by means of a perimeter. In this instrument an object is made to move along a quadrant of a circle, divided into degrees, and rotating on one of its extremities (at which is the fixing-point), so that the quadrant can be placed in each radius of a hemisphere of which the patient's eye is at the centre. The result is recorded on a chart in which are concentric circles divided by radii, at intervals of ten or more degrees. The field does not extend equally in all directions, being limited above by the projecting eyebrow, and on the inner side by the nose. In the diagrams here given only that part of the chart is shown which is included within the average normal field, and for the sake of simplicity only the radii and concentric circles at 30° distance are represented, although many more were employed in taking the fields.

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* The reader will find a full account of them in 'Medical Ophthalmoscopy,' 3rd ed.

It is important, in testing vision, to know that in conditions of functional feebleness of the nervous system, such as hysteria and so-called neurasthenia, the sensibility of the peripheral parts of the retina lessens considerably after attention has been directed to them for a short time, so that even normal fields become smaller when taken several times consecutively, the reduction being confined to the part tested. This effect is still more marked when there is a pathological contraction. It seems to be produced, not in the retina, but at the centre, because, if one part of the field is tested for a short time and becomes narrower in consequence, the same narrowing is found to have taken place in the corresponding part of the field of the other eye.*

It is necessary, in a complete examination, to test the field for colours as well as for white, since defects may be found in the colour-fields where none exist for white. Small pieces of coloured paper may be employed in the same way as a white object. The normal fields for colours are smaller than those for white, and vary for each colour; beyond their limit the object can still be seen, but appears grey instead of coloured. The fields for red and green are the smallest, and undergo most narrowing in morbid states; it is therefore generally necessary to test only these fields. If the power of distinguishing objects is lost in any part of the field, it is desirable to ascertain further whether light can still be perceived in the amblyopic region.†

SYMPTOMS.—*Optic Nerve.*—Disease of the nerve impairs or abolishes vision in one eye, and lessens the reflex action of the pupil in proportion to the interference with vision. The ophthalmoscopic appearances may be at first normal, but if the damage is considerable the optic disc slowly passes into a condition of atrophy. This is termed "secondary atrophy" to distinguish it from the "consecutive atrophy" which follows papillitis. When the nerve is damaged by inflammation, it is not uncommon for slight neuritis to be visible within the eye; but this is usually too trifling in degree to account for the loss. The impairment of sight involves acuity of vision, and commonly also the field. A frequent change in the field is concentric contraction, because the peripheral layer of the nerve, near the optic foramen, is most damaged by processes which begin outside it. It is not rare to have an irregular defect, as in Fig. 72, from one of the cases of rheumatic neuritis mentioned on p. 144. In the others the loss of sight was absolute and lasting. In all, visible atrophy of the nerve slowly supervened, without any recognisable papillitis. A central loss is much less common,

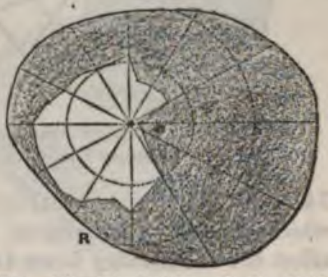

FIG. 72.—Limitation of R. field, greatest in right half, due to orbital neuritis; onset acute, with palsy of ocular muscles and a normal disc; subsequent recovery of the muscles, and slow atrophy of the disc.

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* Schiele, 'Archiv f. Augenheilk.,' Bd. xvi.

† It has been recently asserted that the perception of light depends on a different centre from the perception of form (Wilbrand). It is not yet certain that this is so, but observations on this point are desirable.

and is due to what is termed "axial neuritis," inflammation (or degeneration) in the centre of the nerve. The affected fibres only occupy the centre of the nerve at the back of the orbit; farther forwards they come to (or near) the surface on the temporal side.* This form is met with in "tobacco amblyopia," and it sometimes complicates degenerative diseases of the brain, but does not, strictly speaking, result from them.

Chiasma.—The characteristic symptom of disease of the chiasma is "temporal hemianopia," a loss of the outer half of each field of vision or of part of this half. This temporal hemianopia (shown in Figs. 73, 74, and 75) is the usual symptom, because the morbid processes which damage the chiasma act chiefly on its central portion, and affect the decussating fibres from the nasal half of each retina. In many cases, however, the morbid influence does not remain limited to the central portion, but involves one side of the commissure, or one tract just behind the commissure, or less frequently one optic nerve in front of it. In either case the result is the same; on the side towards which the morbid process extends the non-decussating fibres are involved, and the corresponding eye becomes totally blind, while in the other eye the loss remains limited to the temporal half-field. Often the whole chiasma ultimately suffers, and then the impairment

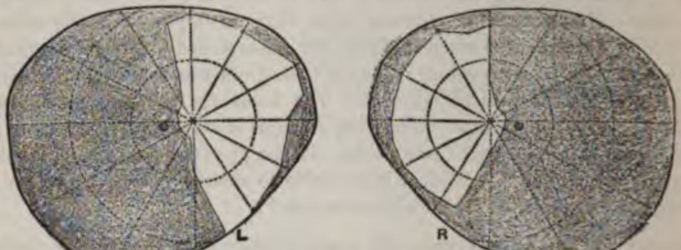

FIG. 73.—Temporal hemianopia, gradual onset; no other symptoms. Not progressive.

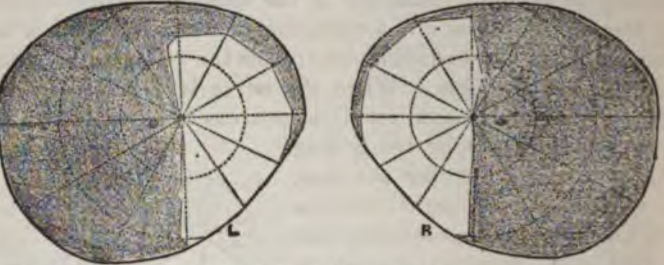

FIG. 74.—Temporal hemianopia without other symptoms than some headache; possibly a tumour of the pituitary body.

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* Nettleship and Edmunds, 'Trans. Oph. Soc.,' vol. i, p. 124; Samelsohn, 'Cent. f. med. Wiss.,' 1880, 418, and 'Trans. Med. Congress, 1881;' Vossius, 'Archiv f. Oph.,' xxviii.

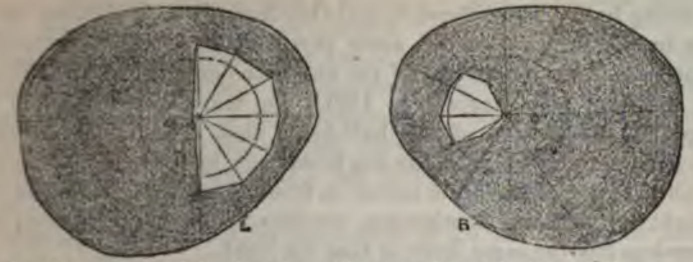

FIG. 75.—Temporal hemianopia, with concentric reduction of the remaining half-fields, greater in the right. In the latter, vision afterwards failed entirely, but the left remained stationary under the treatment, which was based on the assumption that the lesion was chronic inflammation at the chiasma.

of vision progresses through the stages just mentioned to total loss in both eyes.

The rarest of all symptoms of disease of the chiasma is nasal hemianopia. It can only result from a lesion which involves each side of the chiasma, and spares the central portion,—a bilateral external influence, or a symmetrical interstitial affection. It has been due to the pressure of calcified carotids (Knapp). In tabetic atrophy the changes seem sometimes greatest at the chiasma, and irregular nasal hemianopia may be found. The only case that has come under my own observation was of this form. In one eye there was a complete loss of the lower nasal quadrant, extending, however, beyond the middle line; in the other a well-defined amblyopia of corresponding situation (see Fig. 76).

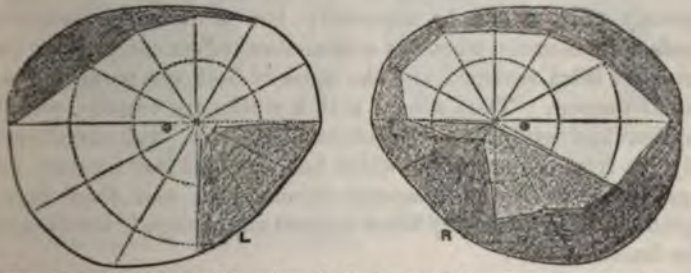

FIG. 76.—Partial nasal hemianopia in a case of optic nerve atrophy. The quadrantic loss in the left field was not absolute; in the right the darker shading indicates the absolute loss.

In the ordinary form of temporal hemianopia the dividing line is seldom exactly vertical. The loss in the two eyes sometimes corresponds closely; more often there is a difference between the two fields. Strict correspondence depends on the strictly central position of the lesion, which seldom obtains in the case of the processes which cause the loss. Sometimes the dividing line appears to pass through the fixing-point, as in Fig. 74; in other cases it diverges to the temporal side of the fixing-point, so as to leave a small area around this within the seeing half. The difference probably depends on individual variations in the decussation; in some persons fibres from the macular region pass in both divisions of the nerve, the crossing and the direct (see "lateral hemianopia," p. 152). In most cases there is no peripheral contraction of the half-fields that remain. When such contraction exists, it is probably due to damage, by inflammation, to the peripheral layer of the optic nerves in front of the chiasma. The loss of sight in temporal hemianopia involves all forms of vision; the colour-loss has the same limit as that for light.

The mode in which temporal hemianopia comes on depends on the nature of the morbid processes which cause it, and these suggest a division into three causal varieties: (1) external compression, as by an advancing growth, or interstitial distension of the third ventricle; (2) interstitial inflammation, primary in character; (3) tabetic atrophy with secondary processes at the decussation. The distinction of the first and second classes is often difficult; because both may be attended by headache, and only the course of the affection may decide its nature. Permanent arrest, under treatment suggested by the hypothesis of inflammation, justifies the diagnosis of this morbid process. In all forms the onset is usually gradual, occasionally rapid, and very rarely actually sudden; the significance of these variations has been already mentioned.

The associations of temporal hemianopia vary according to its cause. Paralysis of some of the adjacent motor nerves to the eyeball-muscles is frequent, when the primary disease is outside the chiasma. Other structures may suffer if a tumour attains a large size. Optic neuritis does not often result from the cause of the hemianopia, certainly less frequently than might be expected; but hemianopia sometimes *succeeds* optic neuritis, when the commissure suffers compression from a distended third ventricle, and the cause of both symptoms is a subtentorial tumour. Thus a man with a stationary tumour, probably syphilitic, and symptoms of internal effusion, had considerable amblyopia from optic neuritis which had subsided into atrophy, when he gradually developed temporal hemianopia, and then became completely and permanently blind without any change in the condition of the fundus oculi.

The course of temporal hemianopia necessarily depends on that of its cause. As we have seen, it may go on to complete blindness; failure of the remaining half-field of one eye may leave the patient only the nasal half of the other field, during the further course of the intra-cranial lesion to the fatal issue which progressive disease usually entails. Sometimes such failure precedes arrest in cases due apparently to a primary inflammation. The damage may be increased by the cicatricial contraction of inflammatory tissue. In such cases the symptoms are not permanently progressive, but it is rare to obtain considerable improvement; although interstitial inflammation is the most probable lesion, we are necessarily ignorant of its precise nature in such cases. It is conceivable that a tumour, arising from the pituitary body, may sometimes cease to increase.

Tract and Hemisphere.—The loss of sight from disease of the visual path behind the chiasma, "homonymous hemianopia," depends on the anatomical arrangement already described. There is an arrest of the impressions from the side of each retina corresponding to the side of the lesion, and hence there is a loss of vision in the opposite half of each field.

It is equally common for the loss to be on the right side and on the left. Of cases collected by Wilbrand, eighty were on the left side and seventy-four on the right. The half-loss varies in its extent. In cases of complete hemianopia it extends up to the vertical middle line (Fig. 77), but the precise character of the dividing line varies considerably in its minute characteristics. The first important difference is that in some cases the line seems to pass through the fixation-point, while more frequently (as in most of the cases figured) the fixation region* is included in the seeing half.† The line of division may occupy the vertical meridian above and below, but about 8° or 10° from the fixation-point it diverges, and curves round this point at about the

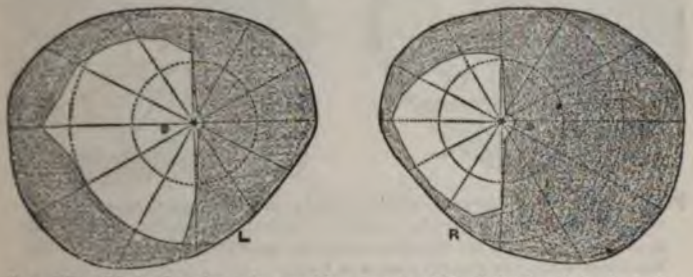

FIG. 77.—Right hemianopia; sudden onset with right hemiplegia and hemianæsthesia, which passed away in a few days, leaving the hemianopia the only permanent symptom.

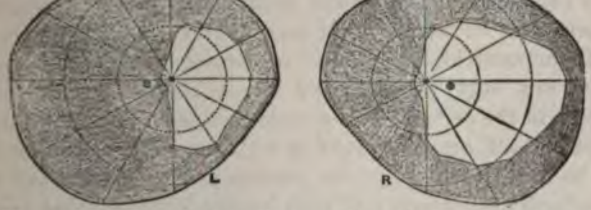

FIG. 78.—Left hemianopia from the case of cerebral tumour shown on p. 145, but probably due, not to the pressure on the tract, but to the interruption of the optic radiation.

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* The region around the fixation-point subserved by the central region of the retina around the macula.

† Wilbrand found that of fifty cases in which the point was specified the line seemed to pass through the fixation-point in twenty-three cases, and to one side of it in thirty-three. See, however, the remarks on page 153.

same distance from it to regain the middle line below (Fig. 77). Sometimes the divergence occurs some distance above the fixing-point (Fig. 78). The central area thus enclosed varies from 3° or 4° to 10° or even 15° radius. In other cases again, the line continues in the meridian, and seems to pass through the fixing-point itself. Rarely it seems to pass through the fixation-point in one eye and beyond it in the other. In a third group the line diverges from the meridian, and when near the level of the fixing-point turns towards it, and seems to reach it, so as to make a re-entrant angle in the outline of the seeing half (Fig. 79). This important difference, the apparent division through the fixing-point or the inclusion of the fixing-point in the seeing half, does not depend on the position of the lesion; each form occurs in cases of hemianopia due to disease of the tract, thalamus, or hemisphere, and we have already seen that each form occurs also in cases of temporal hemianopia from disease of the chiasma. The difference can only be explained by individual variations in the decussation of the nerves. The obvious inclusion of the central region in the seeing half, occurs equally in right- and left-sided hemianopia. It must be due, therefore, to the passage of fibres from the macular region to both optic tracts and both hemispheres, so that this region is not blinded by disease of either tract.*

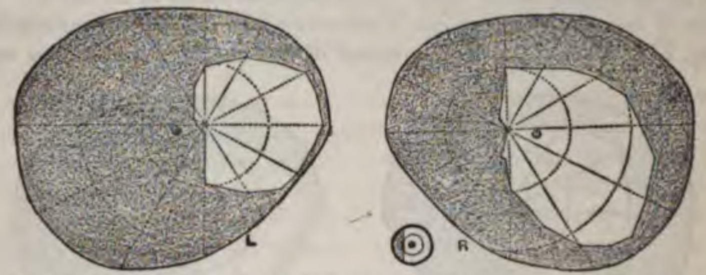

Fig. 79.—Left hemianopia; sudden onset; no other symptoms. In the right eye the loss appears to extend to the fixing-point, but the vertical line in the small figure was drawn by the patient as the limit of the blindness when the central spot was fixed.

Since the distance from the fixing-point at which the dividing line passes varies in different individuals, it appears that there is a corresponding variation in the size of the central area, from which fibres pass to both tracts. This area is very small when the line passes very near the fixing-point, and if it passes through the fixing-point itself there is no double passage of the central fibres, but a division of these as of those from the rest of the retina. Recent careful observations, however, lead me to doubt whether the dividing line ever passes actually through the fixing-point. When it has seemed to do so, I have found, on testing the field with a perimeter and making a more minute examination, that the line passed a little to one side of the actual fixing-point, and did not go through this. In the right field shown in Fig. 79, for instance, the loss, according to the perimeter, seemed to extend quite up to the fixing object; but when the patient fixed a small dot, the area of vision was found to extend beyond it, as shown in the small diagram. If so, the variations in different individuals will depend merely on differences in the area from which the central fibres pass to both tracts, and the apparent difference in kind resolves itself into one of degree.

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* It has been asserted that such passage has been proved by the fact that, in cases of axial neuritis with a central loss of sight, degenerated fibres have been traced into both optic tracts. But they might be so traced, were there a strict division through the macula; the central loss always extends farther from the macula than the area of preserved vision in hemianopia. The passage of fibres from each side of the macula into each tract could not possibly be followed.

Other differences in the dividing line have probably the same origin. In most instances it is somewhat irregular, and in some cases it is oblique above and below. The deviation may be towards one side above and the other side below, so that the general direction of the division is the same, and it may be quite similar in the two eyes.[*] Another difference in the dividing line is also probably due to the anatomical arrangement. The line is usually sharp; good vision suddenly changes to blindness. Sometimes, however, a narrow band of indistinct vision limits the blind half. This must be due to the passage of some fibres from the medial zone by the tract on the same side; they are not numerous enough to give the distinct vision of the macular region, but they are enough to maintain some vision in the middle band.[†] There is no relation between the existence of an amblyopic zone and the seat of the lesion.[‡] All these variations, and also slight differences sometimes to be found between the two fields,

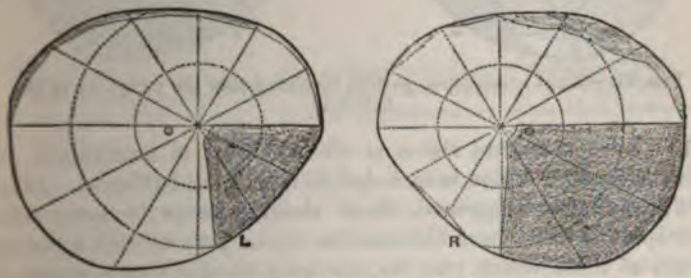

FIG. 80.—Partial left hemianopia involving the lower quadrant. The fields were taken ten years after the onset. The hemianopia was at first complete, and was accompanied by left hemianæsthesia (including the special senses), slight hemiplegia, and optic neuritis. The lesion was probably a syphilitic gumma near the hinder part of the right thalamus. (A chart taken ten years after this one showed precisely the same defect.)

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[*] A well-marked instance of this obliquity is shown in Fig. 15 of my 'Diagnosis of Diseases of the Brain.'

[†] Hirschberg, 'Nägel's Jahresber.,' 1876, p. 79.

[‡] Wilbrand, 'Ueber Hemianopsie,' p. 160. But see Delépine's case, p. 21.

must be due to individual differences in the character of the decussation. When the hemianopia is incomplete the irregularity of outline must be referred to the extent of the lesion.

Hemianopia is not always complete. Only part of a half-field may be lost, but the same part of the field is defective in each eye. Fig. 80 shows a loss of the lower left quadrant in each eye. The lower quadrant seems to be more frequently lost than the upper.* Sometimes the loss is thus partial from the first; more often, as in this case, the hemianopia is at first complete, and vision is regained in some part of the affected half, but remains absent in another part. The loss may extend up to the neighbourhood of the fixing-point, or in slight cases may be confined to the peripheral part of the field (Fig. 81). Such partial loss depends on the partial extent of the disease in the path or centre. It is very rare in disease of the tract, in which all the fibres lie very near together, but has been observed in a case of partial softening (Marchand). In the case shown in Fig. 80 it probably resulted from disease of the optic thalamus or optic radiation near the internal capsule, which was also implicated. Partial hemianopia is often due to disease of the occipital lobe, and upper or lower quadrantic defects are said to be due to disease of the upper or lower parts of the cuneus.

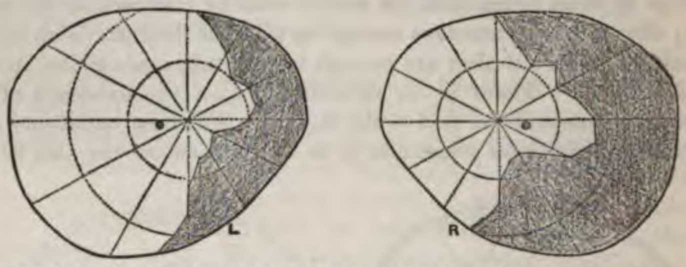

FIG. 81.—Right hemianopia, partial, involving only the periphery of the half-field.

The acuity of central vision is often lowered in hemianopia, even when the fixation region is included in remaining half-field. Indeed, as Wilbrand has suggested, there should always be some central amblyopia; if fibres from the macular region pass by each optic tract, the loss of those which pass by one tract should lessen vision. Of ninety-three cases collected by him, central amblyopia was noted, however, only in fifty-three, and in some of these ophthalmoscopic changes were present. The cases which I have carefully examined show that some diminution of acuity can be detected, if a stringent test is used, even though a cursory examination may reveal no defect.* The number of fibres supplying the macular region is, however, so large that a loss even of one half may have but a slight effect.

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* In the case here figured, and in another figured in 'Diagnosis of Diseases of the Brain,' in which a quadrant was defective, this was the lower one, and the hemianopia was left-sided. Wilbrand's statistics give—left hemianopia, lower quadrant in twenty-one and upper in five cases; right hemianopia, lower quadrant three and upper five—a curious difference. A case is recorded by Hun ('Amer. Journ. Med. Sci.,' Jan., 1887) in which there was defect of the inferior quadrant of left field and superior quadrant (peripheral part) also of left field on both sides. The autopsy revealed atrophy of inferior part of the right crureus.

In many cases of hemianopia the unaffected half-fields have their normal peripheral extent. In other cases there is some peripheral reduction, slight or considerable, and this when there is no peripheral lesion, such as optic neuritis, to which it can be ascribed. The reduction has hardly received the attention it deserves. The half-field is usually smallest in the eye on the side towards which sight is lost, *i.e.* opposite to the lesion; but as this is always the nasal half-field, the peripheral reduction may not seem so great as in the larger temporal half-field of the other eye. The reduction has been supposed to be due to the lesion being in the optic radiation near the thalamus (Wilbrand), but it may be absent even in disease of the occipital cortex.† Its probable significance will be considered presently.

Subjective visual impressions are sometimes described in the blind halves of the fields, usually a faintly luminous fog, but occasionally elaborate, and so definite as to amount to visual hallucinations.

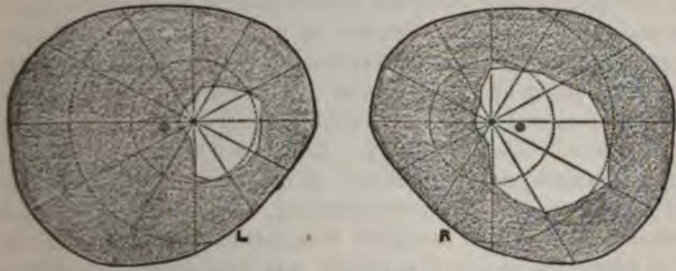

FIG. 82.—Left hemianopia, with a great reduction of remaining half-fields, accompanied by left hemiplegia and recurring left-sided convulsions. The symptoms have now persisted for more than twelve years.

Colour Hemianopia.—In ordinary hemianopia the loss for colours corresponds to that for white. If, however, there is a limitation of the half-fields for white, the colour-fields are also proportionally reduced in size. It is a remarkable fact that there may be hemianopia for colours when there is none for white—"hemiachromatopia." There is no change in the field for objects, but as soon as the vertical line is passed, all colours appear grey. Several cases of this remarkable defect are on record,‡ and it might perhaps be found more frequently if the colour-fields were tested at the bedside in all cases of cerebral disease. The dividing line appears generally to pass through the fixation-point. The symptom probably depends on disease of one part of the occipital lobe, and is proof of a separate centre for colour vision, but the position of this centre is not yet known; Wilbrand suggests that it is in some part of the occipital cortex in front of the apical region, and that impressions pass through the latter to reach the colour centre, since disease of the apex causes complete hemianopia.

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* Of course the possibility that the acuity of vision may not have been normal before the onset of the hemianopia must be borne in mind.

† *E.g.* case of Curschmann, 'Cent. f. Augenh.,' 1879, p. 181.

‡ Bjerrum, 'Cent. f. Augenheilk.,' 1881, p. 471; Samelsohn, 'Cent. f. med. Wiss.,' 1881, Nos. 47 and 50; Eperon, 'Arch. d'Ophth.,' 1884, p. 356; Charpentier, "De la Vision, &c.," 'Thèse de Paris,' 1877; Swanzy, 'Trans. Oph. Soc.,' vol. iii, p.

With the two exceptions just mentioned—the limitation of the remaining half-fields, and the isolated loss for colours—there are no known differences in the character of hemianopia that are due to differences in the seat of the disease in the optic path behind the chiasma.

Double lateral hemianopia, if complete, necessarily involves total loss of sight. It has been known to result from successive lesions in the two occipital lobes, the hemianopia left by the first attack changing to complete amaurosis when the second occurred.* A remarkable case of complete colour-blindness with apoplectiform onset is on record. The acuity and fields of vision were normal. Some colour vision was regained, but there was persistent blindness to red and green.†

Lateral hemianopia is attended by no resulting ophthalmoscopic changes in the early stage. Alterations in the fundus may co-exist, in consequence either of the nature of the cerebral lesion (as papillitis in cases of tumour) or of associated morbid states (as albuminuric retinitis in cerebral hæmorrhage). After some months or years, slight atrophic changes may be visible. Some have thought that pallor could be observed in the corresponding half of each disc. I have not myself been able to recognise this, but have seen the disc on the side on which the temporal half-field was lost become slightly paler than the other. This is explained by the fact that the area of field lost is greater in this eye than in the other, and a correspondingly larger number of fibres have their function arrested.

Lateral hemianopia which has lasted for some weeks in complete degree seldom passes away altogether, and may remain permanent with little diminution. Its persistence for twenty-three years has been observed by Seguin.‡ When a complete hemianopia lessens, it may

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185. Swanzy's case is especially important because there was a reduction of the upper part of the field for white, on the side on which colour vision was lost, which suggests that the centre for colours is adjacent to that part of the half-vision centre in which the upper quadrant is represented. The remaining half-fields for colours were much lessened, and it appears from the chart that there was a slight limitation in that for white.

* Cases recorded by Nothnagel ('Topische Diag. des Gehirnkr.,' p. 389); Calmeil ('Mal. de Cerv.,' vol. ii, p. 411); N. Moore ('St. Barth. Hosp. Rep.,' vol. xv, 1879); Chvostek ('Oest. Zeitschrift' and 'Virchow's Jahresb.,' 1872, vol. ii, p. 49).

† Steffen, 'Arch. f. Ophth.,' xxvii, 1881.

‡ 'Journal of Nervous and Mental Diseases,' Jan., 1886

do so from the medial region towards the periphery, so as to leave a symmetrical peripheral loss, as in Fig. 81, or else from above or below, so as to leave a quadrantic defect. Rarely, however, it clears from the periphery, so as to leave symmetrical scotomata, having one extremity in the central region.*

Hemianopia may remain for a long time unnoticed by the patient, or if he detects a change in vision he is apt to think that there is a loss of sight of one eye. The loss is especially apt to escape notice when a considerable area around the fixing-point is spared, and central vision is thus but little affected. In one case the loss was only discovered when it was noticed by the nurse that the patient at dinner never ate his potatoes, which were always placed on one side of the plate. Hemianopic patients, in circumstances which require a wide range of vision, often carry the head turned a little towards the blind side, so as to bring objects opposite them nearer the centre of the remaining portion of the field. The loss sometimes leads to accidents, especially in those who are unaware of their defect.

Lateral hemianopia is frequently associated with other symptoms of an organic lesion of the brain. Of these, hemiplegia, transient or permanent, is the most common, and is present in at least half the cases.† The loss of power is always on the side of the blindness when the two are the result of a single lesion; the patient is unable to see towards the side that he is unable to move. Hemianæsthesia is also an occasional complication, and speech-defects are sometimes met with in right-sided cases. The significance of these associations will be described in the chapter on "Cerebral Localisation."

As a transient symptom, present only during the early stage of the disease, hemianopia often occurs in severe lesions of one cerebral hemisphere, especially in cerebral hæmorrhage. If the observer's finger is suddenly brought near the eye of the patient, first from one side and then from the other, it will be found that there is a reflex contraction of the orbicularis when the finger approaches from the sound side, and none when it is on the side of the hemiplegia. This seems to show a hemianopic defect, as part of the initial general interference with the functions of the affected hemisphere, although, when the patient has so far recovered as to permit an examination of the field, no defect can be discovered, and the difference in reflex action no longer exists.

Transient functional hemianopia is common as a part of the pheuomena of migraine; and, like other manifestations of that disease, sometimes occurs as an isolated symptom, apart from headache. It is remarkable that hemianopia very seldom occurs as part of the functional disturbance of hysteria, in which another form of sight-defect, presently to be mentioned, is so common. Cases of hysterical hemianopia are on record,* but of a large number of cases of hemianopia that have come under my observation, one only *may* have been of hysterical origin.

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* See a case recorded by Lang and Fitzgerald, 'Trans. Oph. Soc.,' vol. ii, p. 231, where references to other cases will be found.

† The proportion of published cases is one third (59 of 154 cases collected by Wilbrand), but this certainly under-represents the proportion. The more thorough examination of cases now made reveals hemianopia in so many cases of hemiplegia that its presence does not lead to the publication of the case unless this is completed by an autopsy, and it is still probably undiscovered in many cases in which it exists.

Oblique or horizontal hemianopia is seldom met with, and needs further study. It has been mentioned that the dividing line in lateral hemianopia may have an inclination to one side above and to the opposite side below, apparently in consequence of an individual peculiarity in the decussation. It is conceivable that a similar deviation may sometimes give rise to a hemianopia which may fairly be termed oblique. Hemianopia in which the dividing line is horizontal, and an upper or lower half is blind, is very rare.† It may be simulated by a symmetrical lesion of the optic nerves, or may be a compound of double partial hemianopia, in which the two lower quadrants are lost. In the same way a loss of the lower quadrant on the one side, and the upper quadrant on the other side, may conceivably be produced (Wilbrand).

Importance has been attached to the difference, already alluded to, between cases in which the blind half-fields appear to the sufferer merely vacant, or distinctly black, *i. e.* whether there is or is not a sense of entire absence of light in them. It probably depends on the state of the visual centres in the cerebral cortex, especially on the higher centre. In the case of temporal hemianopia, shown in Fig. 73, the patient, a medical practitioner, described the blind halves as appearing, not black, but like a fog; nevertheless he could not even discern a light in them.

A third form of sight-defect from brain disease is what is termed

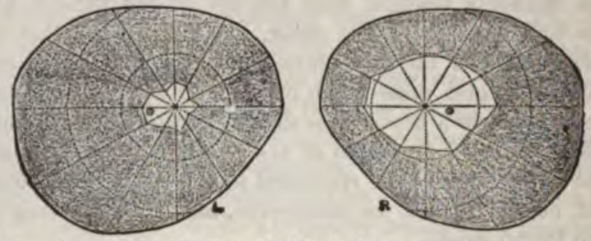

FIG. 83.—Concentric reduction of fields of vision, greater (with considerable amblyopia) in the left eye, accompanied by left hemianæsthesia and slight hemiplegia; sudden onset. The patient was a woman sixty years of age.

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[*] Bonnefoy ("Troubl. de la Vision de l'hystérie," 'Thèse de Paris,' 1874) records some cases observed at the Salpêtrière. Dr. James Anderson ('Ophth. Rev.,' Aug., 1885) has recorded a case in which, along with undoubted symptoms of hysteria, there was complete blindness of the lower halves of the fields, with contraction above and to the inner side.

[†] A case of loss of the lower half of each field is recorded by Anderson, 'Med. Times and Gazette,' 1885, No. 1842.

crossed amblyopia (see p. 22). There is dimness of sight of the eye opposite to the cerebral lesion, and examination of the field shows this to be concentrically reduced in size, in varying degree in different cases. Sometimes it is reduced to a small area, extending only just beyond the blind spot, about 20° around the fixing-point. The colour-fields are also reduced, and disappear altogether when the diminution for white is considerable. The patient does not complain of the sight of the other eye, but if it is tested there is found to be a reduction in this field also, similar, but much slighter in degree, and never involving entire loss of the colour-fields, even of that for red.

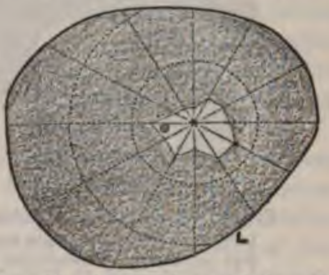

FIG. 84.—Concentric restriction of the field of the left eye (accompanied by amblyopia) in a woman aged fifty.*

This form of sight-defect is similar to that which often occurs in hysteria in association with hemianæsthesia. Pathological evidence of the seat of morbid process is meagre, but is sufficient to make it highly probable that the symptom depends on disease of the cortex of the lower and hinder part of the parietal lobe, the supramarginal and angular convolutions. In the few fatal cases with this loss, the disease has involved this region (see p. 22); while the symptom is not caused by disease of the occipital lobe, or of the temporal lobe, or of the superior parietal lobule, or of the ascending parietal. The facts of disease make it probable, moreover, (1) that the eye of the same side must be represented in slight degree in this centre as well as the opposite eye in chief degree; (2) that partial disease lowers the function of the centre as a whole, and does not cause a partial loss of one part of the field; and (3) that the centre in the opposite hemisphere can supplement, to a considerable degree, that which is destroyed, since the affection of vision usually lessens after a time. Complete destruction of this centre does not cause complete loss of sight of the opposite eye; some vision and a small field remain, and these must be due to the representation of this eye in the other hemisphere, *i. e.* in the hemisphere on the same side as the eye concerned. But disease of this region seldom is complete, because the blood-supply to it is usually shared by both the middle cerebral and the posterior cerebral. Thus each higher visual centre must be connected with both occipital lobes; the connection with that of the opposite hemisphere must be by the fibres of the corpus callosum.

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* The right eye had been removed many years before; the affection of vision was attended by left hemianæsthesia, dulness of all the other special senses on the left side, and severe headache. There was no symptom suggestive of hysteria. Syphilis was probable. The symptoms developed gradually, and increased until iodide of potassium was given, when they became arrested, and they had distinctly lessened when the patient ceased to attend the hospital.

In the theory here advanced to account for crossed amblyopia we may find an explanation of the concentric restriction of the remaining half-fields, often met with in hemianopia. If disease of the occipital lobe, causing half-blindness, extends forward to the higher visual centre, in proportion to the damage to the latter the remaining half-fields will be concentrically reduced, while if the higher centre is completely destroyed, the half-field of the opposite eye will be reduced to very narrow dimensions, vision being subserved only by the other hemisphere. It is possible that a lesion of the white substance beneath this region may have a similar effect. We need more facts before any confident opinion can be expressed regarding the cause or causes of this concentric contraction, but the hypothesis here advanced is supported by the fact that the reduction of the half-field is often much less in the eye on the side of the lesion than in the other eye, just as is the contraction of the whole field when the higher visual centre is alone diseased. In the cases of hemianopia that I have seen, in which the fields were narrowed without peripheral disease of the nerve, and in which there were other localising symptoms, these pointed to disease of the cortex. Thus, in the case shown in Fig. 82, hemiplegia was associated with the hemianopia, and was accompanied by convulsions in the paralysed limbs, commencing locally, and recurring during many years—a sure sign of a cortical lesion. There are few recorded pathological observations bearing on the point, but those that exist confirm the view here expressed.*

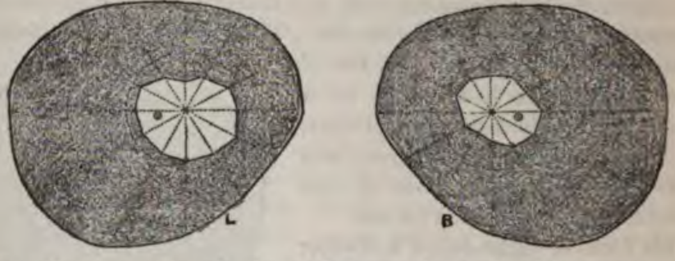

FIG. 85.—Concentric reduction of fields, greater in the right, accompanying right hemiplegia and hemianæsthesia (complete). The onset was preceded by headache and accompanied by convulsion. The patient was a young woman, probably the subject of syphilitic vascular disease. The condition of the paralysed limbs was characteristic of an organic lesion. The fields were taken six weeks after the onset.

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* Thus, in a case of right hemianopia recorded by Forster (Graefe u. Saemisch's 'Handbuch,' Bd. vii, p. 118), in which the peripheral limitation was moderate in degree, the lesion involved the caudate and lenticular nuclei, and also the cortex in a small region in the anterior part of the occipital lobe and posterior part of the angular gyrus. In another case recorded by Hosch ('Klin. Monatsbl. f. Augenkr.,' 1878, p. 281), in which the limitation was considerable, a lesion in the occipital lobe extended from the neighbourhood of the internal capsule up to the grey substance.

Mind-blindness.—The last form of visual defect is the singular condition called "mind-blindness," which has been already briefly mentioned. The term was given by Munk to a condition which he produced in dogs by extirpating parts of the occipital lobes. The animals, while apparently able to see, failed to recognise the nature of objects, such as food placed before them, or to know persons with whom they had previously been familiar. A similar condition has been observed in a few cases of brain disease. Without other mental defect there has seemed to be an entire loss of visual memory; familiar places and faces seemed strange and unfamiliar, and even the nature of familiar objects was not recognised.* The loss necessarily includes that of visual word-symbols.

The structures that subserve the functions lost in mind-blindness are certainly separate from those of the half-vision centres, since hemianopia from disease of the cortex does not necessarily involve this special loss. The centres concerned are probably in front of the half-vision region, either in the anterior part of the occipital lobes or in the posterior part of the parietal lobes. The latter is more probable (see p. 22). The recognition of visual word-symbols is subserved by structures in this region in the left hemisphere, and the loss of this function may, as we have seen, be regarded as partial mind-blindness. We do not know whether complete mind-blindness can be produced by a lesion in one hemisphere, or whether disease of both hemispheres is necessary for the production of the symptom. Its extreme rarity is in favour of a bilateral cause, and experiments on animals suggest the same conclusion. When the condition results from disease of one hemisphere (as in the case figured on p. 23) it is probably transient. If, however, there is a congenital defect or a previous lesion in the higher visual region of one hemisphere, disease limited to the other hemisphere might give rise to permanent mind-blindness.

Although hemianopia does not involve mind-blindness, the two symptoms have been associated. In one case described by Wilbrand, incomplete left hemianopia was associated with a slight defect in the lower parts of the right half-fields, suggestive of a lesion in each hemisphere. Another case is recorded by Bernheim, and is remarkable since considerable and persistent mind-blindness (including word-blindness) accompanied left hemianopia in a left-handed man.†

In this case, however, there were retinal changes. In a case recorded by Wernicke of right hemianopia and peripheral limitation of the fields, the lesion of the occipital cortex extended to the posterior part of the angular gyrus ('Gehirnkr.,' Bd. ii, p. 190). On the other hand, in a case of disease of the white substance recorded by Westphal ('Charité-Annalen,' Bd. vi) without contraction of the field the cortex appeared normal. A cortical lesion did not extend beyond the limits of the occipital lobe in cases without concentric limitation recorded by Curschmann ('Cent. f. Augenh.,' 1879, p. 181).

* Wilbrand ('Die Seelenblindheit,' 1887) has analysed at great length the relations of this symptom to other mental processes.

† Bernheim, 'Revue de Méd.,' 1885, p. 625. This case is consistent with the

In connection with the interference with the higher visual functions of the brain, it may be noted that the area of the cortex, which is apparently related to these functions, is very extensive. In no part of the brain is the difference greater between the brain of the monkey and that of man than in the region of the angular gyrus (in its wider sense, see p. 5). This region in man exceeds in size the whole brain of the monkey.

Diagnosis.—Impairment of sight in one eye without any affection of the other, and with loss of action of the pupil, means disease of one optic nerve. A central scotoma generally means disease of the fibres of the nerve which, at the back of the orbit, occupy the middle of the trunk. Peripheral limitation of the field may be due to damage to the peripheral layer of the nerve; it may probably also be the result of a slight degree of damage to all the fibres of the nerve, which produces most impairment in the periphery of the retina, where vision is normally dull, and is extinguished first by a general reduction of function. A sectorial defect in one eye only means damage to the nerve, considerable in degree, but partial in extent. Amblyopia with concentric reduction of the field, considerable in one eye and slight in the other, may be due to atrophy or to disease of the higher visual centre in one hemisphere In the first the ophthalmoscope reveals the signs of atrophy, and the action of the pupil is lessened; in the second the aspect of the nerve is normal, the pupil contracts perfectly under the influence of light, and the onset is either sudden or attended with other indications of an organic cerebral lesion. In functional (hysterical) amblyopia the symptoms are the same as in disease of the higher visual centre, and the diagnosis depends on other indications of organic or functional disease respectively. In the rare cases of functional disease in which sight is affected in one eye only, the perfect action of the pupil effectually distinguishes the condition from organic disease of the nerve. Total loss of sight of both eyes may be due to chronic atrophy, to damage to the chiasma, or to disease of both tracts or both hemispheres. In all cases the mode of development of the symptoms aids the diagnosis, the symptoms being at first partial; and their character indicates clearly the position of their cause. It is unnecessary to describe in detail the significance of the various combinations of symptoms which sometimes occur in the progress from partial loss to total blindness, since the diagnosis involves only an application of the facts already given in the account of the symptoms.

Temporal hemianopia indicates disease of the chiasma. The combination of complete blindness in one eye, and a loss of the temporal half-field in the other, generally means disease of the chiasma ex-

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suggestion given in the text that unilateral disease only causes persistent mind-blindness when there is a defect in the opposite hemisphere, for left-handedness is often the result of an early lesion or congenital defect of the left hemisphere.

tending to the outer fibres or nerve, and often to the optic tract, on the side on which the blindness is complete.

The diagnosis of the seat and nature of the cause of lateral hemianopia depends on the mode of onset and the associations of the symptom; it is a question of cerebral diagnosis, and will be considered in the chapter on "Localisation." Besides the characters already described, a distinction between disease of the tract, and of the intra-cerebral path and centres, has been found in the action of the pupil when light falls some distance to one side of the macula, called by Wernicke the "hemiopic pupil-reaction."* When the light is thrown on to the blind half of the retina the pupil contracts, as much as if the light is thrown on the seeing half, if the disease is in the hemisphere, but does not contract if the disease is in the tract, because the path to the corpora quadrigemina is then interrupted. The test is of value, although much care is required in its employment, and, with small pupils, the effect may be difficult to discern, and a negative result is often misleading.

PROGNOSIS.—The prognosis depends on the nature of the lesion causing the symptoms. As a general rule it is not good. Considerable defect of sight, due to a lesion of the path or centres, seldom passes away entirely. Hemianopia is especially persistent, and often presents very slight improvement even when the lesion has been stationary for years.

TREATMENT.—The treatment depends on the nature of the morbid process causing the symptoms, and this is described in detail in the account of the several diseases.

FUNCTIONAL AMBLYOPIA AND AMAUROSIS.—*Toxic Amaurosis.*—Bilateral loss of sight, usually complete in degree and extent, but brief in duration, is an occasional result of certain toxic blood-states. It is especially common as a consequence of uræmia. The loss of sight comes on suddenly; sometimes the patient wakes up blind. The pupils are commonly dilated; they may or may not act to light. Other uræmic symptoms, such as convulsions, usually accompany the amaurosis. Sight returns when the state of the blood is improved by treatment. There are no ophthalmoscopic changes related to the amaurosis, but albuminuric retinitis often coincides with it. A similar amaurosis has been met with in cases of lead-poisoning. In poisoning by quinine, and in the acute anæmia that results from loss of blood, sight is sometimes lost, but changes in the retina are commonly met with, although it is not certain that these changes are the cause of the affection of vision. The variations in the action of the pupil in different cases of uræmic amaurosis seem to show that the nerve-elements, on which the blood-state acts, are not always the same. Nerve-cells are more susceptible to arrest of function than nerve-fibres, and therefore we must assume that when the action of the pupil is preserved, the poison acts on the cerebral visual centres; and that when the action of the pupil is lost, the retina itself is rendered insensitive, either alone or together with the cerebral centres. If the amaurosis is sometimes of retinal origin it must be due to a direct action on the nerve-elements, because the absence of any change in the retinal vessels during the loss of sight is well established.

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* Indicated by v. Graefe, and described by Wilbrand, 'Ueber Hemianopsie,' Berlin, 1881, p. 89; Wernicke, 'Fortsch. der Med.,' 1883; and Seguin, 'Journ. Nerv. and Mental Diseases,' 1887, who observes the pupil by light reflected very obliquely from a plane mirror, while throwing in the bright light with an ophthalmoscope mirror.

Migrainous Hemianopia has been already mentioned, and will be described more fully in the account of migraine.

Reflex Amblyopia.—Vision is sometimes impaired in one eye or both by irritation of other nerves, especially by that of the fifth. There is often a concentric contraction of the field of vision, but seldom complete loss of sight. Vision is most affected on the side of the fifth nerve irritated, and the interference is sometimes confined to this eye. The stimulus may be the pain of neuralgia or an irritation of the nerve by organic disease or injury; frequently it is due to a carious tooth. The tooth is generally a molar, although it has been fancied that a recognition of this pathological connection underlies the popular name of "eye-teeth" for the canines. The branch irritated has been generally in the second or first division of the fifth nerve; sometimes it has been a branch of the eyeball itself, as when amblyopia accompanies the photophobia of corneal or conjunctival inflammation. The affection of sight generally subsides when the irritation of the nerve is removed. It does not depend on any visible change in the retina, although vascular dilatation has been seen in some cases. Its mechanism is uncertain, but the most probable explanation is that an inhibitory influence is exerted on the nerve-cells of the retina or on the visual centres in the brain.* At the same time it must be remembered that a similar irritation has been known to set up an actual inflammation in the eyeball. Cases have been recorded in which amaurosis was supposed to be due to the irritation of intestinal worms, but more cannot be said than that this is a possible but certainly very rare cause.

Hysterical Amblyopia.—The characters of the loss of sight that occurs in hysterical hemianæsthesia have been already mentioned, and will be again considered in connection with the other symptoms of this disease. The loss of sight is scarcely ever complete, and when it is considerable in the eye on the anæsthetic side, it is usually distinct, in slighter degree, in the other eye. In rare cases the loss does not conform to this type. Thus I have met with absolute loss of sight in one eye without any affection of the other, and a curious case of paroxysmal complete blindness, associated with abductor palsy of the larynx—both certainly "functional"—is mentioned in the chapter on Hysteria.

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* Compare the case mentioned on p. 167, in which pain in one eye accompanied visual disturbance as the aura of fits due to a tumour of the opposite cerebral hemisphere.

Amblyopia, such as is met with in hysteria, sometimes occurs as an isolated symptom. Although usually transient, it is sometimes of very long duration, and may perhaps be permanent—perpetuated by changes in nutrition which increase to changes in structure. A middle-aged lady who had suffered from various symptoms of functional derangement of the nervous system, cardiac irregularity, vaso-motor swelling of the hands, &c., complained of dimness of sight of the right eye. Acuity of vision was greatly reduced, and the field was contracted to a small area around the fixing-point. The fundus and the action of the pupil were perfectly normal. At no time was there any other sensory loss, or any other symptom to suggest an organic cerebral lesion. The patient was seen occasionally during two years, and in that time the condition of vision and of the eye remained unchanged. Sometimes amaurosis, coming on under conditions suggestive of functional disturbance, goes on to optic nerve atrophy. A lady was always peculiarly sensitive to thunderstorms, although not specially alarmed at them; and she could always foretell a coming storm with remarkable accuracy. During one severe storm she became suddenly blind. There was no suspicion of a lightning-stroke. Vision never returned. I only saw her years after the onset, and there was then simple but complete atrophy of the optic nerves. Such a case may remind us of Charcot's patient with hysterical paraplegia, who recovered, but relapsed under a mental shock, and the paraplegia became perpetuated as lateral sclerosis of the spinal cord, found after death.

It is probable that the seat of the morbid process in functional amblyopia and amaurosis is the cortex of the brain.* The hypothesis of a higher visual centre in each hemisphere enables us to understand its seat more readily than does the theory that the half-vision region constitutes the highest visual centre. Crossed amblyopia, on the latter theory, involves the assumption that there is a combined partial disturbance of both hemispheres, while all the associated symptoms point to the disturbance of one hemisphere only. The nature of the change is a matter of speculation. Arrest of function in the nerve-cells is indicated by the symptoms, and it seems more reasonable to regard this arrest as a primary inhibition than, by assuming vaso-motor spasm, to invoke a derangement of other nerve-cells in the vaso-motor centres. Such a theory only explains the condition by throwing the difficulty further back into the obscurity of the sympathetic. The addition to this theory, favoured in France, that the vaso-motor spasm is the result of uterine irritation, need only be mentioned. The treatment of these functional conditions is that of the general nervous states of which they commonly form part.

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* Priestley Smith ('Ophth. Rev.,' iii, 139) urges that it is retinal. Certainly either hypothesis explains it. When slighter contraction of the other field coexists, the central theory best explains it. Possibly both cortex and retina have some pathological susceptibilities in common, as have other central and peripheral structures. But the pupil-action should decide. He does not mention this.

Irritation Symptoms in the functions of the optic nerves are very rare in organic disease, although common in certain functional maladies, especially in migraine and epilepsy. In migraine, the common form is the zigzag appearance, often coloured, and known as the "fortification spectrum." Similar appearances are occasionally seen without other symptoms of migraine, both by those who are subject to headaches and by others. One patient under my care was troubled with these appearances for years as an isolated symptom. Often the zigzag was arranged in definite relation to some actual visual image, as, for instance, around a plate which was before him. At other times the spectrum was unilateral, and appeared to start from one edge of the field of vision. These phenomena are described more fully in the chapter on Migraine.

In epilepsy visual impressions are very common as the aura of an attack, or as constituting a minor seizure. They are exceedingly varied in character, and are of every degree of elaboration, from a complex visual idea (such as an appearance of an old woman in a red cloak) to a simple flash of light. Stars, or a luminous ball coming nearer or receding, are other forms. Various colours are sometimes seen, especially red or blue; the most common colour is a yellowish red, resembling that of a fire. Simple coloured vision, in which all objects appear coloured, is scarcely ever met with. An apparent increase or reduction in the size of objects seen by the patient is a rare aura, described in the chapter on Epilepsy.* I have known micropsy to precede migrainous headaches. Simple coloured vision, red or blue, lasting for a few minutes, may occur as an isolated symptom in apparently healthy individuals. The cause of the coloured vision occasionally met with in jaundice, and from the administration of santonin, is not known.

Visual hallucinations are common in delirium and insanity, and similar illusions occasionally occur as isolated symptoms in various states of nervous weakness. They are especially common in persons of unstable nervous organisation, in whose families insanity exists. Such patients are often persistently annoyed by spectral visions of various kinds, grinning faces, horrible sights, and the like, when they close their eyes and try to sleep.† Occasionally similar visions disturb such persons in their waking state. Thus one young lady frequently has a distinct vision of a rat or a dog running across the room in which she is sitting. A curious relation has been noted, in a few cases, between such illusions and functional excitation of the optic nerves. We have just seen that a zigzag spectrum may be determined in its position by an actual sense impression; in the cases now under consideration a distinct false vision has existed only when the eyes were open, and has disappeared when they were closed. It would seem that the nerve-cells are only excited to morbid action by functional stimulation. In a curious case, recorded by Hammond,* a woman, during many months, frequently saw before her the image of a man and a woman whenever her eyes were opened, and could at any time produce the vision by hanging her head down. It disappeared when the eyes were closed, and when the right eye was closed one of the figures always disappeared, and the other when the left eye was closed.†

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* In the author's 'Epilepsy and other Convulsive Diseases' a full analysis of these symptoms will be found.

† Hemiopic hallucinations may also occur, *i.e.* the images of objects more or less familiar in the halves of the visual fields. Hemianopia may or may not be present ('Lancet,' 1891, i, 617). See also p. 155.

As a result of organic brain disease, a visual aura has preceded convulsions in several cases of disease of the occipital lobe. In one case of a tumour of the occipital cortex extending as far as the angular gyrus, the aura was a flash of light referred to the left eye and an apparent diminution in the size of objects seen, and sometimes pain in the eyeball.‡ In another case, with visual aura, recorded by Traube, the disease was a cyst in the white substance of the occipital lobe.§

Ocular Hyperæsthesia.—A simple increased sensitiveness of the optic nerve, involving merely an ability to perceive a slighter amount of light or smaller objects than can be perceived by individuals with normal vision, scarcely occurs as an effect of disease. The power of seeing with the naked eye the satellites of Jupiter may be relegated to the category of physiological curiosities. The term "hyperæsthesia" is commonly used in connection with sight to denote the condition in which distress or pain is produced by an amount of light which causes no inconvenience in health. Such intolerance is common in many ocular inflammations or irritation, especially in iritis and keratitis, and is then termed "photophobia." Apart from ocular disease it occurs in weak states of the nervous system, especially in women, and sometimes in association with symptoms of hysteria. In these cases it is termed "retinal hyperæsthesia," but it is not certain that the morbid state is retinal. The symptom was once thought to indicate inflammation of the retina, but in most of these cases the deeper parts of the eye are normal, and actual retinitis, strange to say, may cause no intolerance, nor does this ever result from simple neuritis. Even in health an excessive amount of light is distressing, and in albinoes may act painfully on the retina when that which falls on the eye is not more than can be borne by a normal individual. Optic hyperæsthesia is sometimes accompanied by lessened power of sight, or rather by extreme readiness of exhaustion, so that the field of vision quickly contracts under examination, and use of the eyes rapidly dims the sight. Intolerance of light is always associated with a tendency to protect the eye by closing the lids—"blepharospasm." This may exist, however, without real intolerance of light.

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* 'Journal of Nervous and Mental Disease,' 1885, p. 467.

† A relation which we can understand on the theory that in the higher visual centre the opposite eye is chiefly represented, but not on the current hypothesis that the half-vision relation obtains in the higher as in the lower visual regions.

‡ 'Lancet,' 1879, vol. i, p. 363.

§ 'Gesammt. Beiträge,' Bd. ii, p. 1083. Another case is recorded by Westphal, 'Charité-Annalen,' Bd. vi.

It is remarkable that the diseases that most readily produce photophobia are those of structures that are supplied by the fifth nerve. The reflex consequence, closure of the eyelids, is related to both the fifth and the optic nerves, which guard the eye from mechanical injury and from an excess of light. It is probable that there is a relation between the centres for the two centripetal nerves concerned, so that increased action of the centres of the fifth alters the sensibility of those for vision. At the same time the possibility that light may influence the fifth nerve in the eye cannot be altogether excluded. A curious case has been recorded by Hutchinson in which photophobia was produced by corneal inflammation in a blind eye.

The treatment of intolerance of light is, first, the removal of its cause, whether this is disease in the region of the fifth nerve or defective nutrition of the nervous system; and secondly, the diminution of the surface sensitiveness of the eye, especially by the instillation of cocain, and by cold douches.

MOTOR NERVES OF THE EYEBALL.

(THIRD, FOURTH, AND SIXTH NERVES.)

The motor nerves of the eyeball are the third, fourth, sixth, and sympathetic. The external muscles are supplied by the three former, the internal muscles by the third nerve and the sympathetic. It will be convenient to reserve for special description the affections of the internal muscles, and to consider first the general symptoms of paralysis of the external muscles, the special symptoms of disease of each nerve, and the causes and treatment of such disease.

When there is no muscular contraction the eyeball is in the position of rest, and in this position, under normal circumstances, the line of vision is at right angles to the plane of the face. This is termed its "primary position." Any deviation from the primary position is due to muscular action. Many movements are produced by more than one muscle. Each of the straight muscles turns the eyeball in the direction indicated by its special name, but the superior and inferior recti, in consequence of their origin being nearer the middle line of the body than is their attachment to the eyeball, tend to turn the eyeball inwards and to rotate it, so as to incline the vertical axis of the globe. The upper end of this vertical axis (which it is convenient to indicate by *S.*) is inclined inwards by the superior rectus, outwards by the inferior rectus. This tendency to rotation is counteracted by the simultaneous contraction of the oblique muscles; the inferior, which rotates *S.* outwards, acts with the superior rectus; the superior oblique, which rotates *S.* inwards, acts with the inferior rectus. The oblique muscles also counteract the inward movement of the globe on its vertical axis, caused by the superior and inferior recti, and they also assist the recti in moving the eyeball upwards and downwards, having a slight elevating and depressing action (see diagrams, Figs. 86 and 87). When the eyeball is moved diagonally the lateral rectus acquires a rotating influence, but this is counteracted by the opposite tendency of the associated superior or inferior rectus. In the case of slight inward diagonal movements the rotating tendency of the superior and inferior recti corresponds with that of the lateral muscle concerned, and must therefore be counteracted by an oblique muscle.

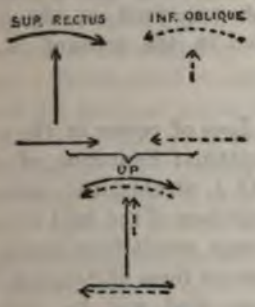

FIG. 86.

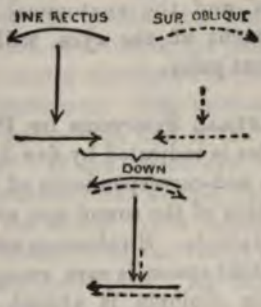

FIG. 87.

FIG. 86.—Action of elevators of right eye; the arrow-heads indicate the direction of movement produced by the muscles named. The curved lines represent the rotation on the antero-posterior axis, the vertical lines that on the transverse axis, and the horizontal lines that on the vertical axis of the eyeball.

FIG. 87.—Action of depressors of right eye. See last fig. and description.

The origin of the eyeball nerves has been already described (p. 52). The tract of grey matter beneath the aqueduct of Sylvius, which gives origin to the third nerves, is continuous behind (*i. e.* below) with that of the nuclei of the fourth nerves, and is connected with the apparently separate nucleus of the sixth by the posterior horizontal fibres. We have as yet no complete evidence of the precise distribution of function in the nucleus of the third nerve in man. The conclusion of

Hensen and Voelcker,* from their experiments on dogs, has been stated at p. 53. A case investigated by Kahler and Pick† suggests that the arrangement of centres is not simply from before back, but that there is also a lateral difference of function. They suggest the accompanying modification of the plan of Hensen and Voelcker. The evidence is strong that the anterior part of the nucleus controls accommodation, and the next the action of the iris to light, and that these functions are subserved by the anterior fasciculi of the roots of the nerve,‡ while a small hæmorrhage behind these centres has paralysed the two internal recti.§

Mid-Line.
Ciliary muscle.
Sphincter iridis.
Lev. palp.
Rect. Int. Rect. sup.
Rect. infer. Obliq. inf.
(Obliq. sup., 4th n.)

The relation between the internal rectus and the opposite external rectus, and the anatomical arrangement concerned in the lateral movement of the eyes, will be described in the account of their conjoint palsy.

GENERAL SYMPTOMS OF PARALYSIS.—Loss of power in the ocular muscles is indicated by five kinds of symptoms: limitation of movement, non-correspondence of visual axes (*i. e.* strabismus), secondary deviation of the sound eye, erroneous projection of the field of vision, and diplopia. Strabismus and diplopia may result from spasm, but persistent spasm is rare except as the common form of "squint," and in this diplopia is absent, for a reason that will be presently mentioned. An abnormal state of the eyeball muscles must not be assumed from the posture during comatose states. In sleep stupor, as in common sleep, the globes may diverge and be directed upwards; one may be higher than the other, and on the patient being roused they may at once assume a normal position. Moreover, a congenital difference of position is far from rare, and may readily mislead; it may also result from old and forgotten injury.

Limitation of Movement.—If a muscle is paralysed, the eyeball cannot be moved so far as normal in the direction of action of that muscle. In complete palsy the defect is absolute; the eyeball cannot be moved beyond mid-position unless slight movement by other muscles is possible. After a time the globe is fixed in the opposite position by contraction of the unopposed antagonist of the paralysed muscle. Thus in paralysis of the external rectus the eyeball cannot be moved outwards, and after a time is turned inwards by the contracture of the internal rectus. If the paralysis is incomplete, movement is deficient in proportion to the amount of palsy, and towards the limit of movement the motion is often jerky ("paralytic nystagmus") from remissions in the contraction, analogous to tremor in a weak limb. When both eyes are open, and acting together, the extent of movement is usually less than when the other eye is closed and the patient uses the affected eye only, and is compelled to strive to "fix" the object with it.

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* 'Arch. f. Ophth.,' Bd. xxiv, 1878, p. 1. See also p. 53.

† 'Prag. Zeitsch. f. Heilk.,' 1881, p. 301, and 'Cent. f. Augenh.,' 1883. The chief evidence is that in two cases of nuclear disease and palsy of some muscles there was a marked difference in the affection of the medial and lateral fibres of some fasciculi. The observed grouping of palsy in disease is seldom significant.

‡ It is not probable that the position of these centres in man is as far forwards beneath the floor of the third ventricle as it is in dogs, and as it has been assumed to be in man by some writers. The nuclei of the third nerves cannot be traced farther forwards than the position of the posterior commissure.

§ Bouchard, 'Rev. de Méd.,' 1891.

Strabismus and Secondary Deviation.—In consequence of the defect in movement, the axes of the two eyes do not correspond in positions that necessitate the action of the paralysed muscle. If a lateral rectus is affected, the axes converge or diverge, producing convergent or divergent "strabismus," which increases, the greater the degree of movement of the sound eye in the direction of action of the paralysed muscle. Paralysis of the external rectus causes convergent strabismus, that of the internal rectus divergent strabismus. The deviation of the axis of the paralysed eye from parallelism with that of the sound eye is termed the "primary deviation." If the sound eye is prevented from seeing the object, and the patient looks at this (is made to "fix" it) only with the affected eye, the sound eye is moved still farther in that direction, and hence the deviation of the visual axes is increased. This is called the "secondary deviation." Its existence and amount may be best ascertained by subsequently covering the paralysed eye, and making the patient "fix" with the unaffected eye, which has then to move back to its former position. The hand, or a piece of paper, may be so placed as to intercept the vision of the one eye, while leaving it exposed to observation. A piece of ground glass placed over the eye answers the same purpose, and permits observation. The occurrence of secondary deviation depends on the fact that normally two muscles which act together are equally innervated for a given movement. If one is weak, and an effort is made to contract it (as in fixing with that eye), the increased innervation influences also the other muscle, and causes an undue contraction. It is as if a rein acted equally on a hard-mouthed and a tender-mouthed horse yoked together; the effort to make the former deviate would cause an excessive deviation of the latter. The secondary deviation is a delicate test for weakness of an ocular muscle, and may reveal its existence when the primary deviation is too slight to be observed. Moreover these deviations afford a distinction from strabismus due to muscular spasm, such as ordinary squint. In the latter the deviation exists in all movements; in paralytic strabismus, only in those movements that call into action the paralysed muscle. In ordinary strabismus, the deviation is the same whether the patient fixes with one eye or with the other; in paralytic strabismus fixation with the normal eye evokes the primary, and that with the affected eye the secondary deviation: these two are similar in direction, but opposite in character, the primary being a defect, and the secondary an excess of movement.

Erroneous Projection.—An interesting and important effect of the increased innervation that causes the secondary deviation, is erroneous projection of the visual field. We judge of the relation of external objects to each other by the relation of their images on the retina; but we judge of their relation to our own body by the position of the eyeball as indicated to us by the innervation we give to the ocular muscles. When there is no muscular effort, and the eyes are at rest in mid-position, we know, to use popular language, that an object at which we are looking is opposite our face; that is, that a line from the centre of the field of vision to the macula lutea is perpendicular to the facial plane. If we turn the eyes towards one side, we know that an object in the middle of the field is to the side of its former position; how far to that side we estimate by the degree of movement of the eyes indicated by the amount of innervation; and if we want to touch the object, the knowledge thus gained enables us to so contract the muscles of the arm as to effect the desired movement with precision. But the increased innervation needed by a weak ocular muscle gives the impression of a greater movement of the eye than has really taken place, and suggests that the objects seen are further from the middle line than they really are. If the patient then attempts to touch an object, the finger goes beyond it. The erroneous projection exists, of course, only in those movements of the eyes for which the weak muscle is necessary. It is greatest when the affected eye is used alone; it disappears altogether when this is closed. The erroneous projection is always in the direction of action of the affected muscle. The knowledge of the relation of external objects to the body, gained from the contraction (*i. e.* innervation) of the eyeball muscles, is one of the most important sources of guidance to the centres that regulate the muscular contractions for maintaining the equilibrium of the body. The erroneous projection of the field destroys the harmony between the visual impressions and the others that are correct; the resulting discord and its consequences may affect consciousness as the sensation of "giddiness." This giddiness depending on paralysis of an ocular muscle, is termed "ocular vertigo."

Double Vision.—We have seen that the field of vision of the paralysed eye is erroneously projected. Each object in the field is referred to a position other than that which it actually occupies, and corresponding to the degree of ineffectual innervation. But if the patient looks with both eyes, the field of the unaffected eye, being normally projected, does not correspond with the field of the affected eye; the images formed in the two eyes are mentally referred to different positions; objects are seen double. The patient is conscious of the doubling only of that object the image of which is formed at the macula lutea of one eye or the other, that is of the object which is fixed by one eye, for which the eye is accommodated, and to which the attention is directed. The image formed in the unaffected eye, being referred to its correct position, is termed the "true image," the other is termed the "false image." The distance between the two images depends on the amount of the error of projection, and corresponds to the degree of deviation of the eyeball from the position which it should occupy with that amount of innervation. In consequence of this deviation of the two visual axes, the retinal image is formed at the macula lutea only in one eye (the fixing eye). In the other eye it falls upon a part of the retina away from the macula, at a distance from it which corresponds with the degree of deviation, and therefore with the amount of erroneous projection. It was formerly thought that the circumstance that the two images are formed upon non-corresponding parts of the two retinæ affords an adequate explanation of the diplopia. But the diplopia which occurs in secondary contracture of a muscle cannot be explained by the simple theory of non-correspondence of retinal areas.

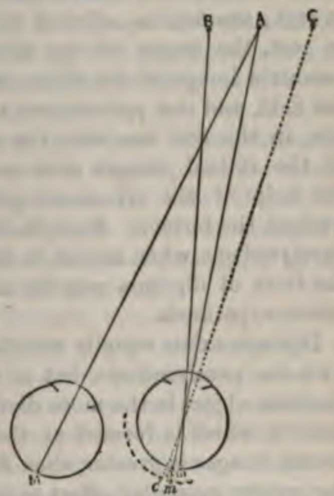

FIG. 88.—Diagram to illustrate the production of diplopia by the erroneous projection. In consequence of weakness of the right external rectus the image of A formed at the macula, M, of the left eye, falls within the macula of the right eye at a. But the effort corresponds to that necessary to bring the macula to *m* (in the broken arc). Hence B, whose image is at m, seems to occupy the position of A, and the image formed at a to the left of m is projected to the other side of A, and seems to occupy the position C, as if it were formed at *c* to the left of *m*.

muscle—the right external rectus, for instance—is paralysed, and when all the muscles are at rest and each eye should be directed straight forwards, the right eye is turned inwards by the permanent shortening of the unopposed internal rectus. The image of an object opposite the eye is formed on the macula lutea of the left eye, but to the inner side of the macula lutea of the right. That this is no reason why there should be diplopia will be evident if we suppose for a moment that the patient has only the right eye, that this is normal and voluntarily turned to the left. It is clear that the object in the position supposed will be seen (not distinctly, because its image falls on an excentric part of the retina) in its actual place. So, too, if we suppose the patient to have only the left eye, and that this is directed towards the object; this will also be seen in its normal place. Thus there is no reason in the mere deviation of the eyes, and the non-correspondence in position of the retinal images, why the object should be seen in this eye in an erroneous position,—why there should be double vision. The explanation is that in the case supposed—of a normal eye being turned to the left—the field is mentally projected to the left, in correspondence with the degree of active muscular innervation; the centre of the field lies thus to the left, and the object straight in front is referred to its proper excentric position in the field. But in the case of the contractured eye all the muscles are at rest; the field is referred to the position normally corresponding to rest, the centre of the field is projected directly in front, the excentric image of the object is referred to an excentric position in the field, and the patient sees the object in a false position with this eye, in the real one with the other. Thus the non-correspondence of the retinal images does not explain the diplopia, except with the help of the erroneous projection. The latter does explain it without the former. Nevertheless, if there is no contracture, and the paralysed eye, when at rest, is in mid-position (as in recent paralysis), the facts of diplopia may be explained, and its laws formulated, on either hypothesis.

Diplopia exists equally whether the patient fixes with the normal or with the paralysed eye, but in the former case the true, in the latter the false object is the more distinct, that image being always the more distinct which is formed at the macula. The distance between the double images is greater when fixation is by the paralysed eye, because the greater muscular effort results in a wider error of projection; of this greater effort the secondary deviation of the non-paralysed eye is evidence. As Graefe has pointed out, if the patient moves his finger from the fixing (paralysed) eye to the apparent image, in trying to touch it, the line taken by the finger corresponds in direction with the visual line of the other (secondary deviating) eye; each represents the excessive amount of muscular innervation, and the degree of excess is a measure of the erroneous projection of the field of the paralysed eye.

In recent paralysis, diplopia exists only when the eyeball is in a position that needs the action of the paralysed muscle. In old cases with secondary contracture and permanent deviation, diplopia may occur during rest, or even during other movements. The distance between the double images increases, the farther the object is moved in the line of action of the affected muscle. As a rule it is the false image which appears to move from the other, and the direction of its movement is that of the action of the muscle.

The false image may be on the same side of the other as the eye by which it is seen (*homonymous or simple diplopia*), or it may be on the other side (*crossed diplopia*). When the muscle which is paralysed is an abductor, and the eyes therefore converge, the diplopia is simple; when the muscle is an adductor, and the eyes diverge in paralysis, the diplopia is crossed. This depends on the fact that, in the former case (of convergent strabismus from loss of abduction), the false image is formed on the side of the retina towards the sound eye, and is therefore projected on the other side of the real object. In the case of divergent strabismus from paralysis of an adductor the false image is formed on the side of the retina farthest from the sound eye, and is therefore projected on the side of the real object towards the sound eye. The student may remember the relation by the rule that when the visual lines (prolonged ocular axes) cross, the diplopia is *not* crossed.

In diagonal positions of the eyeballs the two images are not parallel; the false one appears inclined. On the theory that the diplopia is due to the non-correspondence of the parts of the retinæ on which the images are formed, the obliquity is explained by the fact that an abnormal rotation occurs in these diagonal positions. This rotation is due to the action of the oblique muscle, which should prevent rotation by opposing this tendency of the paralysed muscle. The obliquity is explicable also on the theory that the diplopia is due to erroneous projection: as v. Graefe has shown, the same principles apply to an abnormal rotation as to a defect in lateral movement.

The total field of vision which is brought into view by various movements of the eyes, the head being still, may be represented as a circle, the centre of which is at the fixing-point when the eyes are at rest, while the radii represent the various movements, horizontal, vertical, and diagonal. The circle is termed the "motor field." In paralysis of one muscle without secondary contracture there is diplopia only in one part of this field, that corresponding to the movement of the muscle. Thus in paralysis of the right external rectus there is diplopia in the right half of the field. The line which separates the area of single or double vision varies in direction with each muscle, and corresponds with the position of the axis of the eyeball around which the globe moves when the muscle is in action; but the correspondence is seldom exact, on account of the co-operation of other muscles in certain positions. To lessen the double vision the patient often holds his head in such a position as to call the paralysed muscle into action as little as possible. The movement of the head is on an axis which corresponds to the line of separation of single from double vision, and thus to the axis on which the eyeball is moved by the affected muscle.

Patients are not always conscious of double vision. If the muscular weakness is slight the two images may nearly correspond, and the result may be an indistinct outline to the apparently single image. Again, if the images are far apart the less distinct excentric image may be neglected, and the patient may be conscious only of that which is seen by the normal eye. This is generally the case in old palsies. The detection of diplopia is facilitated by placing a red glass before one eye, preferably the sound eye, because the more distinct image seen with this eye is thus rendered less preponderant. A strip of white paper is then held up in various parts of the motor field; the patient looks at it without moving his head: one image is seen red, the other white. The two are thus more easily distinguished, and their relative position can be ascertained. If the red glass alone does not at first suffice, the view from each eye may be alternately obstructed (as by the hand held before the eye); and if there is double vision the position of the object will seem to alter as it changes from red to white. Such obstruction will, alone, often elicit unnoticed diplopia.

In employing diplopia in diagnosis we must first prove that it is not monocular, by ascertaining that it ceases when one eye only is used.[*] We then learn by the coloured glass whether the double vision is simple or crossed, whether the two images are on the same level or not, whether parallel, and the direction in which movement of the object increases the distance between them. When the images are side by side, and the diplopia is simple, the paralysed muscle is of the eye on the side towards which movement increases the distance between the images. If the diplopia is crossed, it is a muscle of the other eye. If the images are one above the other, the paralysed eye is that of which the image ascends from the other in looking up, descends in looking down. If there is secondary contracture of the antagonist of the paralysed muscle, diplopia exists through the whole range of movement in the plane of action of the muscles, *i. e.* during the action not only of the paralysed muscle, but also of its antagonist; but the distance between the images during movement remains the same on the side of the contractured antagonist, while it alters on the side of the paralysed muscle. It must be remembered that the vertical action of the superior and inferior recti is greatest in abduction of the eye, and that during adduction the oblique muscles also elevate and depress. Vertical movements are therefore in the line of action of two sets of muscles, and to discriminate between them we must move the object up and down both during adduction and abduction. Vertical diplopia in abduction is due to the superior or inferior rectus; if in adduction only, it may be due to paralysis of an oblique muscle.

Aid in diagnosis is also to be obtained by prisms, on the principle that a prism so placed that its base corresponds with the direction of action of the paralysed muscle, increases the distance between the double images; one in the opposite position tends to blend them. They may also be brought together by a prism placed before the unaffected eye in the position in which before the affected eye the diplopia is increased. Fusion by a prism before the affected eye removes the erroneous projection; fusion by a prism before the normal eye brings the projection of this field into the same error as the other.

Paralysis of Individual Muscles.—We may now consider the symptoms of paralysis of each muscle. It must be remembered that more than one muscle is often affected, and the symptoms are correspondingly combined; and that in diseases of the central nervous system, palsy and spasm are sometimes so associated as to render an exact diagnosis difficult and occasionally impossible. This difficulty is often due to the circumstance that a given palsy was preceded by some other derangement of movement, as another slight paralysis and secondary contracture.

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[*] The cause of monocular diplopia is still unknown. It is sometimes well marked when there is no discoverable abnormality in the refraction or in the fundus of the eye.

The paralyses described and figured are of the muscles of the right eye, except that of the external rectus, which is of the left eye for comparison with the paralysis of the opposite internal rectus.[*]

External Rectus, Left (Fig. 89).—The defect of movement is outwards, with convergent strabismus; secondary deviation is by the opposite internal rectus. The diplopia is simple, in the left half of the motor field; the two images are on the same level in the horizontal movement, and the distance between them increases as the object is moved to the left. In diagonal positions the false image is inclined, the images diverging from the horizontal plane. Towards the limit of movement there are often slight spasmodic rotatory movements by the unopposed oblique muscle. The head is inclined to the left.

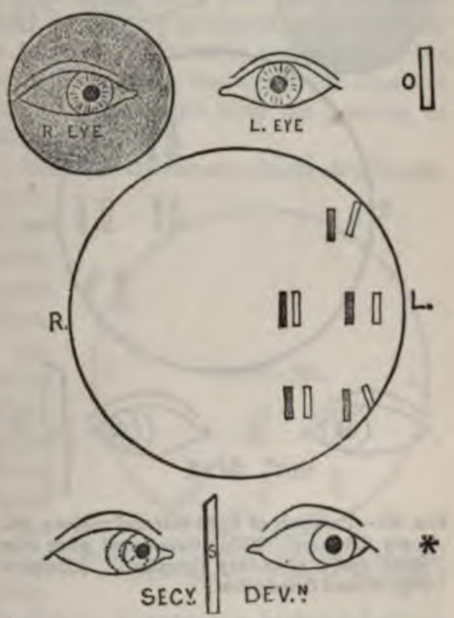

FIG. 89.—Paralysis of the left external rectus; coloured glass over right eye; primary deviation on looking towards an object (O) on the left; position of double images; secondary deviation of the right eye when the screen, *s*, obstructing the fixation of * by this eye, compels fixation by the weak muscle. When the screens are removed, the right eye, in fixing, moves back to the position of the dotted outline of the cornea. (In the diagram of diplopia the highest white [false] image should have been represented a little lower than the true image.)

Internal Rectus, Right (Fig. 90).—The defect of movement is inwards; the secondary deviation is by the opposite external rectus; the strabismus is divergent. Diplopia is crossed, and exists in the left half of the motor field.

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[*] The diagrams of double vision are after those of Woinow ('Das Verh. der Doppelbilder,' Wien, 1870), modified a little by actual observation. The objects are figured as described by a patient when they are held between him and the observer. If figured as seen by the patient, the position of the object is reversed.

In diagonal movements the images diverge towards the horizontal plane. The head is inclined towards the left.

Superior Rectus **(Fig. 91).—Movement is defective upwards, especially upwards and outwards, and in attempting the movement the eyeball is rotated, the upper end of the vertical meridian outwards, by the inferior oblique. Secondary deviation is by the opposite superior rectus. The diplopia is crossed and exists in the upper half of the motor field; the images are one above the other, the upper image being the false one, and receding from the other as the object is moved upwards. The false image is inclined. The difference in height is greatest in abduction, while the inclination is greatest in adduction. These differences are due to the influence of the inferior oblique, which rotates as well as elevates. The head is held backwards and inclined towards the sound side.**

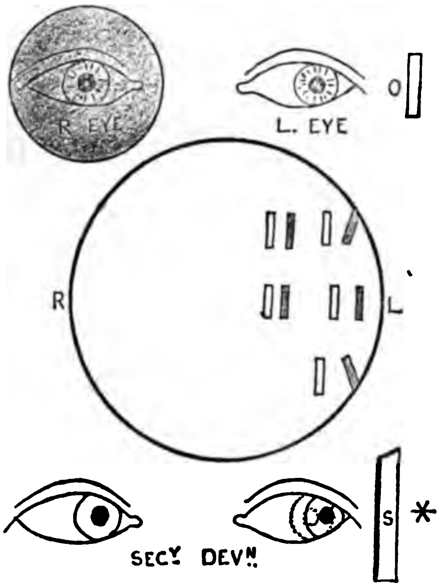

Fig. 90.—Paralysis of right internal rectus; primary deviation; double vision (red glass over right eye); secondary deviation. (Compare Fig. 89 and description.)

Inferior Rectus **(Fig. 92).—Movement is defective downwards, especially downwards and outwards, because in the**

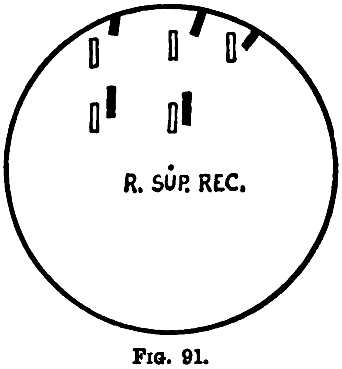

Fig. 91.

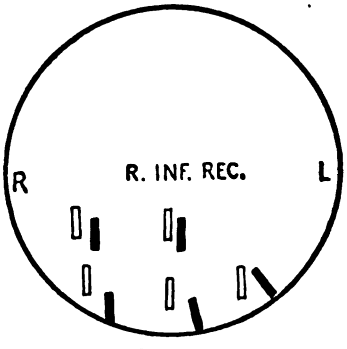

Fig. 92.

Fig. 91.—Diplopia in paralysis of right superior rectus. (The black image is that of affected eye.)

Fig. 92.—Diplopia in paralysis of the right inferior rectus. (Right image black.)

downward and inward movement the superior oblique is best able to supplement the paralysed rectus. The upper end of the vertical meridian is inclined inwards by the oblique, from the loss of the rotating action of the inferior rectus. Diplopia exists in the lower half of the field, especially in the outer portion; it is crossed, the images being near together laterally, but the false image is below the other, and recedes with the downward movement. The two images (if the object is held vertically) diverge from the horizontal plane. The difference of height is greatest on looking inwards; that of obliquity on looking outwards. Secondary deviation is produced by the opposite inferior rectus. The head is inclined downwards and towards the affected side. In an attempt to look down, the upper lid does not descend, but remains raised in correspondence with the position of the eye (see Fig. 94, and "Facial Paralysis").

Superior Oblique (Fig. 93).—Defect of movement chiefly downwards and inwards, since in this position the muscle exerts most depression, but it is often scarcely recognisable. Strabismus exists only below the horizontal plane, and is convergent by the unopposed inferior rectus, causing deviation inwards. Secondary deviation is chiefly by the opposite superior oblique and inferior rectus, &c., turning the eye down and in. Diplopia exists in the inner lower part of the motor field; it is simple; the false image is the lower, and the images diverge towards the horizontal plane. The difference in height is greatest in adduction, the obliquity of the false image is greatest in abduction. The lateral distance is greatest in the middle line, and lessens when the object is moved either inwards or outwards. The head is inclined forwards and towards the sound side. The diplopia is chiefly noticed when the patient has to look down, as in descending stairs, which appear double. The detection of this palsy is difficult, and the diplopia is the most important indication; if the other eye cannot be moved downwards the detection of the palsy may be impossible.

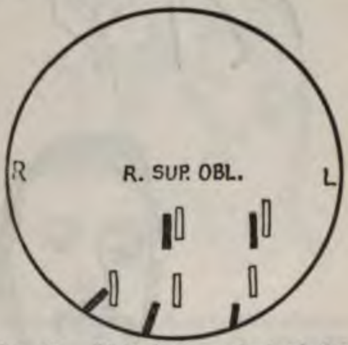

FIG. 93.—Diplopia in paralysis of right superior oblique. (Right image black.)

Inferior Oblique.—Defect of movement inwards and upwards, the effect being the converse of that of paralysis of the superior oblique. The secondary deviation is inwards and upwards by the inferior oblique and superior rectus, &c., of the sound eye. Diplopia exists in the upper inner part of the field; it is simple, the false image above the other, especially in adduction, and oblique (the images converging towards the horizontal plane), especially in abduction. The head is inclined backwards and towards the affected side.

Of isolated palsies, that of the external rectus is incomparably the most frequent, and the order or frequency of 145 cases was found by A. Graefe to be—external rectus 105, superior oblique 52, inferior rectus 10, superior rectus 9, internal rectus 4, inferior oblique 2. The frequency of isolated palsies of the first two muscles is due to their separate innervation. In the estimate, the frequency of loss of some of the associated palsies is probably not included. They were not then adequately recognised.

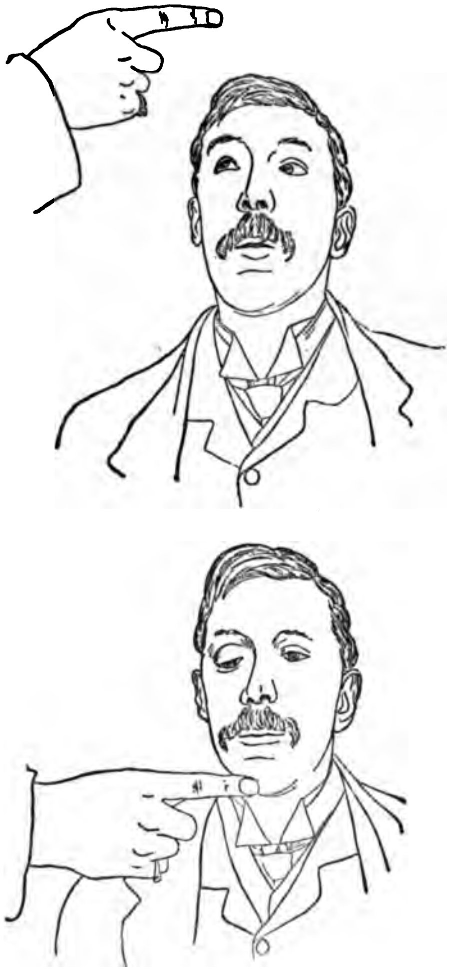

Fig. 94.—Partial paralysis of left third nerve. Defective movement upwards, from weakness of the superior rectus, and downwards, from weakness of inferior rectus.

AFFECTIONS OF SPECIAL NERVES.

Sixth Nerve, Abducens Oculi.—Only one muscle, the external rectus, being supplied by the sixth nerve, the disease of the nerve causes only paralysis of this muscle. As the figures given above show, it is a frequent ocular palsy, perhaps because the nerves have so long and exposed a course.

*Third Nerve.**—When the whole of the third nerve is affected, all the external muscles of the eye are paralysed, with the exception of the superior oblique and external rectus; the levator palpebræ, sphincter iridis, and ciliary muscle are also affected. The upper eyelid droops and cannot be raised; the eye can be moved only outwards, and a little downwards and inwards by the superior oblique. The pupil is of medium size, and does not contract; power of accommodation is lost.

*** The third nerve is commonly termed in Germany the "oculo-motorius," a name which has the grave practical inconvenience of preventing the use of the term "oculo-motor" in its exact and much-needed general significance.**

In an attempt to raise the eyelid the frontal muscle contracts strongly (see next page). After a time the deviation outwards of the eye increases, in consequence of contracture in the external rectus, and the pupil becomes larger than it was at first by a similar contracture in the radiating fibres.

Paralysis of the third nerve is often partial; the various muscles supplied by it are affected in different degrees, and some may escape altogether. The muscles to the eyeball may be involved, and the levator may escape, but the paralysis of the former is then seldom complete. The levator and superior rectus may suffer apart or together; in bilateral nuclear disease they are generally paralysed together. The external muscles may suffer, and those within the globe may be unaffected; the converse relation is only seen in central cases.

Both third nerves may be affected, but it is rare for both to be paralysed completely, as in the case shown in Fig. 95. There is then complete double ptosis, and if the lids are raised the eyeballs are seen to be turned outwards, and to be almost immovable. In most cases of bilateral disease of these nerves the paralysis is partial. In some cases the distribution of the palsy is determined by functional association; thus each internal rectus may be affected and no other muscles: such cases are central and nuclear in nature, involving this nerve merely because its functions are so extensive; they will be separately considered.

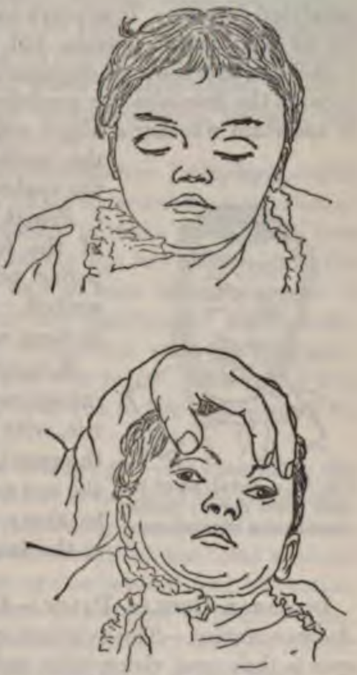

FIG. 95.—Paralysis of both third nerves in a child two years old. Complete double ptosis, and divergence of both eyes by the external recti when the eyelids are raised. The child was of a tubercular family, and presented also double optic neuritis and weakness of the limbs of both sides. No other cranial nerves were affected. The probable cause of the symptoms was a tubercular tumour in the interpeduncular space. Under tonic treatment all the symptoms passed away, and a year later the child appeared quite well.

Ptosis.—The drooping of the eyelid may be complete, so that it cannot be raised, or incomplete, being raised to a certain point but not higher. There is usually a conspicuous over-action in the corresponding half of the frontalis, so that the eyebrow is always higher than it should be. The frontalis normally acts with the levator when the eyes are directed much above the horizontal plane. This is an instance of secondary over-action in an associated muscle, analogous to the secondary deviation in an ocular muscle (p. 171). The frontalis responds to the increased innervation, which has no influence on the paralysed levator. It appears as if the patient were trying to raise the lid by the frontal muscle, but this appearance is, at least chiefly, fallacious, and the real explanation is that just given. But in some persons the frontalis can produce a slight elevation of the lid; hence to ascertain whether a slight voluntary elevation of the lid is due to the levator or frontalis, it is necessary to fix the eyebrow by pressing it against the bone.

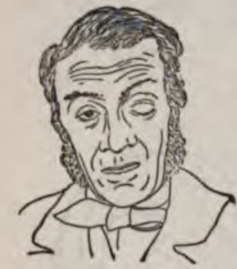
FIG. 96.—Paralysis of the left third nerve; ptosis; over-action of frontalis.

Fourth Nerve.—The symptom of paralysis of the fourth nerve is paralysis of the superior oblique, which has been already described. It is seldom met with alone, except in cases of nuclear disease.*

Sympathetic.—Disease of the sympathetic causes paralysis of the radiating fibres of the iris, with consequent loss of the dilatation of the pupil. There are also slight prominence of the eye and slight ptosis, supposed to be due to atony of the involuntary fibres of Müller in the fascia of the orbit.

INTERNAL OCULAR PALSY.—*Ciliary Muscle: Cycloplegia; Loss of Accommodation.*—The evidence of this in normal and hypermetropic eyes is that near vision fails and distant vision remains good, while near vision can be restored by a convex glass. Usually, though not invariably, the contraction of the iris which normally occurs on accommodation is absent also. In myopia and presbyopia the absence of the power of accommodation makes little difference to sight, and the diagnosis of the condition is difficult. Accommodation depends on the third nerve, and is lost in complete, and often in partial, damage to the trunk of the nerve, and in disease of the anterior fibres by which it arises, or of the anterior part of its nucleus. The path from the lenticular nerve is through the ganglion and ciliary nerves. Loss of accommodation in one eye only, without the affection of other branches of the third nerve, is possibly due to disease of this ganglion or of the nervous ganglia within the eye, but we have no evidence at present of the effect of disease of these structures.† Bilateral cycloplegia, occurring alone, usually depends on disease of the centres in the nuclei of the third nerves. It occurs in dipththeritic paralysis, of which it is one of the earliest and most constant symptoms. It occurs also as an associated symptom in degenerative disease of the spinal cord, especially in tabes. Its relation to such disease is similar to that of the loss of the reflex action, immediately to be described.

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* See Remak, 'Neur. Cent.,' 1888, p. 5.

† See Hosch, 'Neur. Cent.,' 1889, p. 592.

Iris: Iridoplegia.—The pupil may be unduly large (*mydriasis*), from palsy of the sphincter (third nerve) or spasm of the radiating fibres (sympathetic), or it may be unduly small (*myosis*) from the opposite conditions. The iris has three actions, two reflex and one associated: (1) Reflex contraction of the sphincter on exposure of the eye to light. (2) Reflex dilatation by the radiating fibres on stimulation of some cutaneous nerve. (3) Contraction on accommodation, usually, but not necessarily, associated with convergence.

(1) *Loss of the Associated Action: Accommodation Iridoplegia.*—The pupil does not lessen in size when an effort to accommodate is made. To examine this action it is only necessary to make the patient look first at a distant and then at a near object; the two should be in the same line of vision, so as to avoid any change in the amount of light that enters the eye. There is usually paralysis of accommodation, but the ciliary muscle may be competent, and yet the associated action of the iris may be lost, and *vice versâ*. This loss is less common than is that of reflex action; it is due to the same causes as cycloplegia.

(2) *Loss of the Light Reflex: Reflex Iridoplegia.*—In examining the action of the iris to light, care must be taken to test each eye separately, keeping the other eye covered. Light entering one eye acts on both pupils, and the contraction of each is the result of the total amount of light that enters both eyes. If one eye be covered and the action of the other pupil noted, it will be found to contract still more when the first eye is uncovered. A good light is important; a slight action, distinct on a bright day, is often imperceptible in a dull light. If artificial light is used, it is best to make the patient look at an object in a distant dark part of the room, and then bring a light suddenly in front of the eye. If the patient looks at a near light, he will accommodate for it, and the pupil may then contract when there is no action to light. Hence, if the light is looked at, it must be at least four feet from the patient, and should be bright. To avoid the accommodation it is desirable, if a light is brought in front of the eye, that the other eye should be shielded from the light but not closed, so that, by continuing to fix a distant object, accommodation may be kept relaxed. Reflection of light into the eye by an ophthalmoscopic mirror is not a good test, because the light falls only on a very small area, and may miss the macula, the most sensitive part. Reflex iridoplegia is usually double; when one-sided, as in a case recorded by Eales,* its cause is usually exceptional. But I have observed the loss to be unilateral in tabes, although this is very rare.

The path through which this reflex action is produced is the optic nerve, both optic tracts, probably the corpora quadrigemina, the anterior part of the nucleus of the third nerve behind the centre for accommodation, probably the second fasciculus of origin of the third nerve, its trunk, the ciliary ganglion, and the ciliary nerves.

* 'Ophth. Rev.,' ii, p. 225.

(3) *Loss of the Skin Reflex: Cutaneous Iridoplegia.*—The dilatation on cutaneous stimulation occurs in most persons under normal circumstances. The skin of the neck is a convenient place for the stimulation. The faradic current applied with a wire brush, so as to produce a sharp painful sensation, has been usually employed, but in most persons the prick of a needle or pin, of a quill point, and even a pinch, is sufficient.* The motor path for this action lies in the cervical sympathetic, and the fibres connecting this with the cord at the lowest part of the cervical region. The centre on which it depends is said to be beneath the corpora quadrigemina, to the outer side of that for the light reflex. If so, both motor and sensory paths must traverse the cervical region of the spinal cord.

Each reaction is lost when disease interrupts its path or damages the centre on which it depends. Thus the light reflex is impaired or lost in disease of either the optic nerve (including the retina) or in disease of the trunk of the third nerve. Disease of one optic tract does not lessen the action, unless the light falls only on the blind half of the retina (see p. 163), no doubt because the fibres from the most sensitive (central) region of each retina pass by both optic tracts, and so disease of one does not abolish the reflex. So, too, the skin reflex is lost in disease of the cervical sympathetic, and in some affections of the cervical spinal cord, especially in those that impair sensibility. In any case we infer the seat of the disease from the other symptoms that are associated with the loss.

When the loss of reflex action occurs without such accompanying symptoms, and is thus isolated, so far as immediate associations are concerned, it is generally due to disease of the centres, degenerative in character.† It occurs in association with disease of the spinal cord, chiefly with locomotor ataxy, in which it is a common and early symptom. The loss is frequent also in general paralysis of the insane, and is occasionally met with in other degenerative diseases of less definite type. It may occur also without other nerve symptoms. In most of the cases I have seen, in which it existed alone, the patients have had constitutional syphilis many years before, and the same statement is true of at least one disease (locomotor ataxy) with which the symptom is often associated.

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* In some persons in whom the reflex action is readily produced, it occurs on slighter cutaneous impressions. It may occur, for instance, when the palm of the hand is tickled, as schoolboys know who pretend to dilate the pupil by an act of the will.

† This has not yet been demonstrated, but hardly admits of doubt because the loss of reflex action occurs under the same conditions as another affection to be presently described, progressive paralysis of the external muscles, the central and degenerative nature of which has been proved.

The frequency of loss of the light reflex without loss of the associated action of the iris was first pointed out by Argyll-Robertson. The fact that loss of reflex dilatation is usually associated with that of reflex contraction was discovered by Erb.

The two palsies are often, but not always, conjoined. There may be loss of reflex action to light, when the pupil still dilates on stimulation of the skin. The pupils are often small, reduced to two, one and a half, or one millimetre in diameter. The association of these small pupils with spinal disease was noticed before the more significant loss of the light reflex was discovered, and the condition was called "spinal myosis." But the pupils are not always small; they may be three, four, or five millimetres in diameter, although there is no trace of reaction to light. I think that it will generally be found that when the pupils are not small the skin reflex persists, and when they are small it is lost. The small pupils may be the result of the loss of tone in the radiating fibres accompanying the loss of the skin-reflex, and preserved when the pupils are large. In cases of disease of the third nerve the ciliary muscle is paralysed as well as the iris. When the reflex loss is due to central degeneration the ciliary muscle is usually unaffected, and the pupils contract during accommodation. In some cases, however, there is also cycloplegia. All the internal muscles of the eyeball are then paralysed, a condition which we shall again consider.

COMPOUND OCULAR PALSIES.—*Loss of Convergence and of Accommodation.*—The power of accommodation may be lost although the eyes can still be converged, but in many cases the two actions, habitually associated, are lost together. The internal recti may contract in a normal manner in lateral movements of the eyes, but cannot be made to contract together. Doubtless there is a separate centre for the converging action, as we shall presently see there is for the action of one with the opposite external rectus in lateral movements. The former must be closely connected with the accommodation centre, since we only converge when we accommodate. A remarkable example of this loss in a child has been published by Eales.* There was absolute loss of convergence, of accommodation, and of the associated action of the iris, while all other movements of the eyes, and the reflex action of the pupil, were perfect. The loss developed gradually without recognisable cause, and, after lasting for about a year, passed away.

Paralysis of the upward movement of the eyes has often been observed in cases of central disease, associated with paralysis of the levators. It may result from disease of the posterior part of the third nerve nuclei (Kahler and Pick). When due to such a focal lesion it is usually unilateral. It is possible that there is also a higher centre, disease of which may paralyse the upward movement without the lid, since this isolated symptom may be met with. I have recorded one such case in which the symptom was well marked.†

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* 'Trans. Ophth. Soc.,' iv, 1884, p. 300, and oral communication.

† 'Trans. Ophth. Soc.,' i, 81, p. 117. The patient has died since the account of her case was published. A very small tumour was found in the middle line

Paralysis of both upward and downward movements of the eyes, without impairment of the lateral movements, has also been observed, and is probably also due to a lesion in the same position. The paralysis has been bilateral and accompanied with ptosis. But it has also been caused by disease of the cerebral hemisphere, situated in the corpus striatum and optic thalamus.*

Paralysis of Lateral Movement: Conjugate Deviation of the Eyes.—The conjugate deviation of the eyes, towards the side of a lesion in the cerebral hemisphere, the result of a loss of the power of moving them to the other side, has been already described (p. 78). The deviation is occasionally due to spasm, and is then from the side of the cerebral lesion. We infer to which of these two mechanisms it is due, by the associated symptoms in the limbs, which are paralysed and flaccid when the eyes deviate from them, rigid with spasm when the deviation is towards them, and from the side of the lesion. These are merely an extension to the eyes of the effects of the disease manifest in the limbs. Their occurrence and degree seem to be related to the seat of the cerebral lesion. But in disease of the pons the condition is reversed. A paralysing lesion on one side causes a loss of the movement of both eyes towards that side.† The seat of the disease that has this effect is the tegmental region of the pons, at or above the nucleus of the sixth,‡ and the facts show that the path for this combined movement descends in the same side of the pons as that towards which the eyes are moved, and that the movement is effected through the nucleus of the sixth. The sixth nucleus must therefore act on the the opposite third nucleus and internal rectus, and the path by which the influence is exerted is almost certainly the posterior horizontal fibres. It has even been thought that through these fibres, those of the third nerve for the internal rectus actually arise from

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behind the posterior quadrigeminal bodies, damaging these slightly, the velum, and the adjacent part of the inferior vermiform process of the cerebellum.

It must be remembered that disease of the nerves or their roots may chance to affect only the fibres for the superior recti. This was apparently the case in a patient with an interpeduncular syphiloma (Thomsen, 'Berlin. Gesellsch. f. Psych.,' June 7th, 1886). One superior rectus was more affected than the other, a character that is probably of diagnostic importance.

* Wernicke, 'Berlin. kl. Wochenschr.,' 1876, p. 394, and 1878, p. 154. A similar loss, associated with hemianopia, was present in a case recorded by Lang and Fitzgerald, 'Trans. Ophth. Soc.,' vol. ii, p. 230.

† Much attention has been lately given to this palsy. The most important writings on the subject are those of Broadbent, 'Med. Times and Gaz.,' 1872, vol. i; Duval and Laborde, 'Journ. de l'Anat. et Phys.,' 1879; Bernhardt, 'Gehirngeschwülste;' Wernicke, 'Gehirnkrankheiten,' Bd. i; Mierzejewski and Rosenbach, 'Neurolog. Centralbl.,' 1885, p. 363; and Bleuler, 'Deut. Arch. f. kl. Med.,' 1886, Bde. xxxvii and xxxviii, in whose paper abstracts of most writings on the subject will be found, and an important case by Hughes Bennet ('Brain,' 1889), in which there was permanent conjugate deviation of head and eyes, the result of a patch of softening limited to the sixth nucleus. Power of convergence was retained.

‡ Very seldom below, and then reaching up to the nucleus. See note on next page.

the sixth nucleus; this assumption is opposed by many facts, and it seems that the horizontal fibres merely connect the two nuclei.[*] Other signs of disease of the pons are often present, paralysis either of the facial or of the fifth nerve. Nystagmus is often associated (see p. 207).[†]

Since the movement is excited from the opposite cerebral hemisphere, the path must cross the middle line above the middle of the pons, and it probably does so at the corpora quadrigemina. If so the arrangement must be that shown in Fig. 97. We do not know, however, whether the pons contains merely the path to the sixth nucleus, or whether there is a special centre for the movement distinct from this nucleus. The latter is probable on account of the relation of the movement to sensory impressions, especially to those of the auditory nerve, combined with the fact that a movement of the head is often assöciated with that of the eyes, so that a connection of the auditory nerve with the sixth nucleus would scarcely suffice. The connections of the superior olivary body, according to Bechterew, are precisely

FIG. 97.

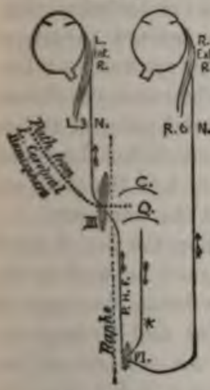

FIG. 98.

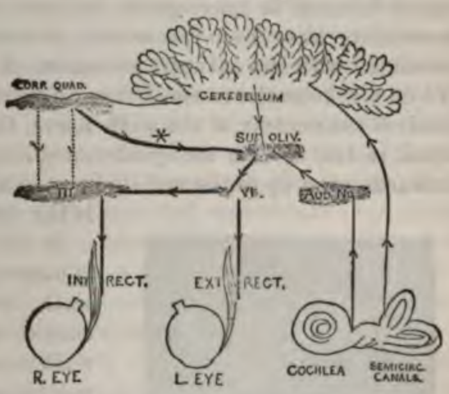

FIG. 97.—Diagram of the mechanism for the associated lateral movement.

FIG. 98.—Diagram of probable relation of the superior olivary body to the associated lateral movement. The asterisk in each figure indicates the downward path.

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[*] The evidence is that the internal rectus is not always totally paralysed; it has been known to act in convergence although not in the lateral movement, and also inwards, in a normal manner, if the other eye was covered, although there was no movement when the eye with the paralysed external rectus was uncovered. Moreover, when the sixth nucleus has been totally destroyed, no degenerated fibres could be found in the opposite third nerve.

[†] Experiments suggest that deviation towards the side of a lesion of the pons means irritation and imperfect destruction. The opposite deviation follows subsidence of the irritation, each being attended by nystagmus (Laborde, 'Gaz. méd. de Paris,' 1878)

those that such a centre would have. By some fibres of the fillet, which degenerate downwards, this body is connected with the corpora quadrigemina, and fibres pass between it and the sixth nucleus, the auditory nuclei, the cerebellum, and the lateral column of the spinal cord; the last may subserve the movement of the head.* If this hypothesis is correct, the mechanism must be such as is shown, in side view, in the diagram Fig. 98. The arrow-heads indicate the direction of conduction; and the thicker lines indicate the path by which the movement is excited by the will.

According as the disease does or does not involve the nucleus of the sixth nerve, the symptoms present some variation. If the disease is above the nucleus, there is a loss of the power of moving both eyes beyond the middle line towards the side of the lesion. If they are moved towards the other side, they can be moved back as far as the middle line, but no further. In most cases the other eye cannot be moved inwards in convergence or alone, but in a few instances these movements have been preserved, although the inward movement associated with the outward movement of the other eye has been lost. We do not know the cause of this difference. If, however, the disease involves the nucleus of the sixth nerve, there is total palsy of the external rectus, so that the eye deviates inwards and cannot be moved outwards, even up to the middle line. The condition of the other eye is the same as in the first case.

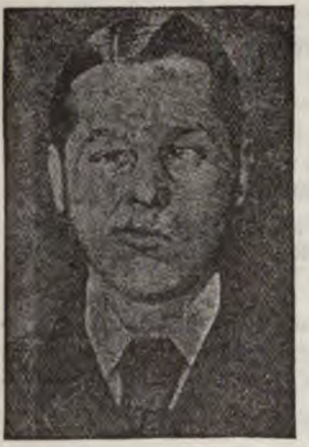

FIG. 99.—Complete paralysis of the left sixth and facial nerves (with inability to move the right eye towards the left) from an acute lesion on the left side of the pons. (From a photograph.)

In this condition the facial nerve is usually paralysed as well as the sixth, its fibres being damaged as they course round and through the sixth nucleus. An instance of this palsy is represented by Fig. 99. The onset was sudden, and slight hemiplegia was present at first. The left sixth and facial nerves were completely paralysed; the left eye deviated inwards, and no outward movement was possible; the right eye could not be moved inwards; the facial muscles presented the reaction of degeneration. If a lesion in the pons damages the fibres of the sixth nerve away from their nucleus, the paralysis of the external rectus is complete but exists alone; there is no deficiency of movement of the opposite internal rectus. The lesion that causes these symptoms may be either a chronic process, such as a small tumour, or an acute lesion, hæmorrhage, or, more frequently, softening. Other symptoms of disease on one side of the pons are often present, especially in the region of the fifth nerve on the side of the lesion, sometimes in the limbs on the opposite side.

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* In one recorded case the lesion was below the level of the sixth nucleus, the lower end of this nucleus being near the highest part of the lesion. It is probable that the superior olivary body was involved (Senator, 'Berl. Psych. Gesellsch.,' July 9th, 1883).

Total Ophthalmoplegia.—Paralysis of all the muscles of both eyes, internal and external, is a very rare consequence of disease at the neighbourhood of the orbital fissure and optic foramen on each side, either in the orbit or within the skull, such as thrombosis in each cavernous sinus.* But it is met with chiefly in cases of nuclear disease, and will be described in the account of this variety.

CAUSES AND CAUSAL VARIETIES.—Paralysis of the ocular muscles may be due to disease of the nerves in the orbit or at the base of the brain, to lesions of the fibres of origin between the surface of the brain and the nerve-nuclei, and to disease of the nuclei themselves. A defect in certain ocular movements may also be produced by disease of the cortex of the brain and of the path from the cortex to the nuclei, but the loss that is usually so produced is that of the conjugate lateral movement of both eyes, already described in the account of hemiplegia, with slight ptosis on the side opposite to the lesion. It is possible, however, that a defect of associated movements (*e. g.* of the upward or downward movement) is also sometimes due to disease of the hemisphere, but our knowledge of this cause is imperfect. Of the remaining lesions those of the nuclei (constituting the important group of nuclear palsies) will be separately described.

Injury is a common cause, and may damage the nerves in any part of their course, especially at the orbital fissure or in the orbit, but sometimes at their origin. Some of the roots of one third nerve have been found torn.† One eye is usually alone affected. The injury is usually a blow on the side of the head, which causes an extravasation and immediate paralysis, or excites an inflammation and palsy after a few days. All or only some of the nerves may suffer. Recovery is slow and often imperfect. Sometimes, indeed, there is a late extension, perhaps by processes outside the nerves, or cicatricial contraction of adjacent tissue. Thus a blow on the right temple was followed at once by palsy of all parts of this third nerve. Slow improvement was followed by a relapse, with palsy of the external rectus.

Neuritis in the orbit or at the base of the brain, involving the trunks or branches, is not rare. It may be syphilitic in either situation. In the orbit it is sometimes primary and "rheumatic," but it is occasionally secondary to adjacent cellulitis. Within the skull it is generally secondary to meningitis. The nerves in the orbit are damaged by slow pressure only in rare cases of orbital growths, but within the skull they often suffer compression by growths or aneurisms. All the nerves to one eye may be compressed in the wall of the cavernous sinus by thrombosis in it, by an aneurism of the internal carotid, or by an adjacent growth,* and both third nerves may be damaged by a growth between the cerebral peduncles, or by an aneurism of the posterior cerebral artery. Tumours of the nerves themselves are rare; neuromata are almost unknown, but nodular syphilitic growths sometimes occur, and may be symmetrical—situated, for instance, on the two sixth nerves near their origin (Barlow). The root-fibres suffer, in their deep course, from various lesions of the brain,—hæmorrhage, softening, tumours, and islets of disseminated sclerosis. Such lesions, involving the sixth or third nerve-fibres, often also implicate the motor tract, and there results hemiplegia associated with palsy of one of these nerves. In consequence of the decussation of the motor tract below the origin of the nerve, the two symptoms are always on the opposite sides, "alternate hemiplegia;" if on the same side there must be two lesions. Very rarely a small focus of softening in the crus may damage only some of the roots of the third nerve.† Paralysis of all the ocular muscles has been caused by the fumes of charcoal, by lead, and that of the third nerve has been met with in Graves' disease.‡ Other toxæmic states, as that from alcohol, may have a similar effect. In some, the mechanism is a peripheral neuritis, but an action on the centre is more frequent.

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* Coupland, 'Trans. Oph. Soc.,' vol. v.

† Hutchinson, Astley Cooper Prize, May 1st, 1865.

Diphtheritic Paralysis.—A rare cause of palsy of the external muscles is diphtheria. The external rectus has been occasionally affected, and both internal recti have been weakened in association with the loss of accommodation so common in this disease. In one case all the muscles supplied by one third nerve were affected (Vadelot), and in two others there was paralysis of all the external muscles of both eyes.§

Tabetic Paralysis.—In locomotor ataxy it is very common for some of the ocular muscles to be weakened. The loss of power is often transient, and then may recur; sometimes it is permanent. Each form may occur at any stage of the disease, and is sometimes an early symptom, even preceding those in the limbs. The external rectus, or the levator and superior rectus, are the muscles most frequently affected; occasionally all the muscles supplied by the third nerve suffer. Loss of the light-reflex is extremely common, and other internal palsies are not infrequent. External palsy is generally on one side only, but sometimes both eyes are affected (see Figure, vol. i, p. 408). We have scarcely any evidence at present of the cause of these palsies, but the fact that total nuclear palsy is sometimes met with makes it probable that these also are often of nuclear origin. The peripheral nerve degeneration of tabes has not been found in the ocular nerves, and its limitation elsewhere to the sensory fibres renders its occurrence in these nerves improbable. It must be remembered that many subjects of tabes have had constitutional syphilis, and that a true syphilitic ocular palsy may co-exist with locomotor ataxy.

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* Many instances of such palsy are on record. A typical one is recorded by Nettleship, 'Trans. Ophth. Soc.,' vol. i, p. 186.

† Kahler and Pick, 'Arch. f. Psych.,' x, 334. It has been termed "fascicular palsy." "Root palsy," internal or external, would be a better name.

‡ Knapp, Buzzard, Finlayson.

§ Uhthoff, 'Neur. Centralbl.,' 1885, p. 125; and Mendel, ib., p. 128.

Rheumatic Paralysis.—Affections of the ocular muscles due to exposure to cold are regarded as due to rheumatic neuritis. They are always one-sided, and commonly involve a single nerve, or a single branch, or two contiguous branches, such as those to the levator palpebræ and superior rectus. Rarely all the nerves of one orbit are involved, including the optic nerve (see p. 144). The onset of the disease is often attended with pain about the orbit. The occurrence of a rheumatic neuritis does not rest on pathological evidence, but is nevertheless highly probable. In two of the cases in which all the nerves of the orbit were affected the patient had previously suffered from facial neuritis due to cold. In one recorded case, in which swelling of the lids seemed to prove that the disease was orbital, the internal muscles escaped, on account, it is supposed, of the more central position of their nerves.*

Hæmorrhage.—Paralysis of an ocular nerve may be apparently the result of hæmorrhage into the sheath, compressing the fibres. In a case of paralysis of one fourth nerve, the onset was quite sudden during an attack of vomiting. No other cause could be traced. Hæmorrhage into the sheath of the third nerve, near its origin, has preceded, by a few days, meningeal hæmorrhage.† Other cranial nerves may be suddenly paralysed in like manner. This mechanism is probably not uncommon and may depend on a special arrangement of the blood-vessels. The cases come within the general law that an organic lesion of actually sudden onset is always vascular, due to the rupture or occlusion of a vessel. An onset during strain renders rupture scarcely questionable.

Syphilitic Paralysis.—From the list of causes already given it is evident that syphilis may produce palsy of ocular muscles in several ways. The nerves may be the seat of isolated syphilitic inflammation or of a gumma; they may be involved in syphilitic meningitis or compressed by a syphilitic growth outside them. They may also be damaged by an aneurism of syphilitic origin. A young man, a few years after primary syphilis, became affected with palsy of one third nerve, which did not yield to antisyphilitic (or any) treatment. After some months he was seized with apoplexy and died, probably from the rupture of an aneurism of the posterior cerebral artery. Lastly, syphilis seems to predispose to degeneration of the nuclei of the nerves. Thus the mere fact that paralysis is due to syphilis is only the first step in the diagnostic problem of the nature of the disease.

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* Möbius, 'Cent. f. Nervenh.,' 1886, p. 516. The symptoms followed exposure to cold.

† Goldscheider 'Charité-Ann.,' 1892.

Relapsing Palsy.—One form of paralysis of the third nerves, which occurs chiefly in the subjects of syphilis, presents a peculiar tendency to relapse and persist. One third nerve becomes paralysed, improves under treatment, and then, perhaps while the treatment is being continued, the other third nerve suffers, and afterwards the affection of the first returns. An instance of the palsy is shown in Fig. 100. Syphilis was contracted four years previously, but the sudden onset of the palsy of the left nerve occurred during a severe cold. The patient was treated with iodide and mercury, but the course of the paralysis was that described above, and the permanent condition was that the left eye could only be moved outwards and a little upwards. There was extreme ptosis of the left eye and slight ptosis of the right. The internal muscles were also paralysed. In another case, similar in course but less complete, the interval after the syphilis was thirty years, so that its influence is doubtful. The nature of the lesion in these cases is uncertain. The course and limitation of the symptoms seems inconsistent with nuclear disease.

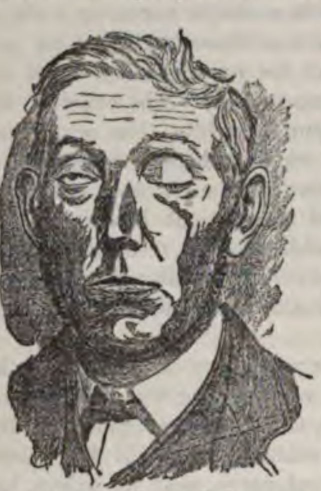

FIG. 100.—Double ptosis from paralysis of the third nerves, complete on the left side, partial on the right. Over-action of frontales, greater on the side of the complete palsy.

Recurring or Periodical Palsy.—The last variety to be described is a rare form of transient palsy, usually on one side only, which comes on at intervals during many years.* It has been miscalled "relapsing palsy," but typical attacks are recurrences, not relapses, and, in most instances, have been periodical. Both sexes suffer, but women are more subject to the disease than men. It often dates from early childhood, and has been known to begin at eleven months,† at fifteen months,‡ at five years of age, and later;§ it continues up to at least middle life. An attack may occur at the same time each year, or at intervals of about six months. A much longer interval usually separates the early attacks. Sometimes the intervals have been long and irregular, amounting to several years,‖

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* Cases have been recorded in this country by Saundby, 'Lancet,' 1882, ii, 345, and 1885, i, 57; and by Snell, 'Trans. Ophth. Soc.,' v, 193.

† Möbius, 'Neur. Cent.,' 1884, p. 307.

‡ Snell, loc. cit.

§ Thomsen, 'Charité-Annalen,' 1885, p. 562.

‖ Five to nine years in a case recorded by Camuset, 'L'Union méd.,' 1876, p. 906.

while in other cases they have been short, and attacks have even occurred at each menstrual period.[*] As a rule the paroxysm begins with severe pain in the eye, and often with headache and vomiting. These symptoms last two or three days; the palsy may accompany them or may come on as they lessen. In many cases only the third nerve has been affected, but in some the external rectus was also involved. The palsy may be complete or incomplete; the internal muscles are often, but not always, affected; there is usually ptosis. The loss of power lasts for a few days or weeks, and then gradually passes away. When the attacks are frequent they are usually brief. In many cases a slight defect of power has persisted during the intervals. In one case there was concentric contraction of both fields of vision, greatest in the paralysed eye, and varying in degree in proportion to the motor palsy (Thomsen). The attacks may last for several weeks, from three to eight; and, as a general rule, when the intervals are long, so are the attacks. When the paroxysms recur frequently—as, for instance, every month—they last for a few days only. A child of three and a half, with some indications of congenital syphilis, had ptosis of the right eye. Four months later it had ptosis of the left eye, and a second attack in the same eye ten months afterwards. Each time the attack lasted a few days, and passed off without leaving any weakness. In several cases the headache was unilateral. In patients subject to the affection, an attack has been brought on by mental shock.

The nature of this disease is mysterious. It has been compared to migraine, and is perhaps more closely allied to this than to any other disease. Charcot has termed it "migraine ophthalmique," and in one case there was a strong family history of migraine (Snell). But the long duration of the attacks, the motor character of the chief symptoms, their long duration, and the occasional persistence of slight defect of movement, are marked differences from purely migrainous and neuralgic affections.[†] It has been suggested that the cause is organic disease with periods of activity, but this theory is scarcely tenable. Vaso-motor disturbance has, of course, been invoked to explain it, but this is only putting the difficulty farther back. The few post-mortem examinations so far obtained do not clear up the mystery. With permanent weakness there have been changes in the peripheral nerves and not in the nuclei. A woman died of phthisis at thirty who had suffered, since childhood, from periodical attacks of palsy of the left third nerve. This appeared grey, and its roots were surrounded by small grey granulations which contained tubercular bacilli, which did not extend into the crus. Some fatty degeneration was found in the muscles supplied by it, the other muscles being normal.* In other three examinations the lesion was peripheral and the nuclei intact.†

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[*] Hasner, 'Prag. med. Wochenschr.,' 1883, No. 10.

[†] A case recorded by Buzzard ('Clin. Lect.,' p. 164) is perhaps a connecting link between these cases and pure neuralgias. A woman had been subject for many years to fortnightly paroxysms of neuralgia of the first division of the fifth nerve, and for two years before she came under observation each attack was *followed* by partial palsy of the third nerve lasting for a few days. Complete palsy has occurred during each attack of migraine (Anderson, 'Trans. Oph. Soc.,' 1892).

In a case recorded by Pflüger there were periodical attacks of paralysis of variable seat; of the left third and facial nerves at eighteen, of the nerves on the right side at twenty, of the left third nerve again at twenty-two, and six months later of the left sixth and facial. Each attack lasted from one to two months, and was preceded by pain, which before the last was about the left mastoid process.

NUCLEAR OCULAR PALSY.—Disease of their nuclei is a frequent cause of paralysis of the eye-muscles, internal and external, and certain forms may be due to disease of the corpora quadrigemina which does not penetrate the nuclei. The symptoms vary much according to the position and character of the lesion, which may be various, and either acute or chronic; but they are, as a rule, bilateral, except when there is the isolated affection of the sixth nucleus, already considered in the account of such conjugate palsy.‡

The chief forms of nuclear palsy may be classified according to the character of the symptoms and probable nature of the morbid process. The most common is (1) chronic nuclear palsy, due usually to degenerative processes, very rarely to such chronic disease as a tumour. A rare form is (2) acute nuclear palsy, in which the symptoms come on in a few days or a few weeks. The onset suggests inflammation, and the form has been termed (by Wernicke) "polio-encephalitis superior,"§ from its analogy with polio-myelitis, and to distinguish it from a similar affection of the lower nuclei in the medulla, "polio-encephalitis inferior." But it may apparently also be excited by toxic influences. (3) In *sudden nuclear palsy* the onset occupies a few minutes, or at most an hour or two. These cases are often miscalled "acute," but this term is needed for class 2. Such an onset always indicates a vascular lesion, which probably is the common cause of this form.

The characteristic feature of all forms is the paralysis of muscles of both eyes, internal or external, symmetrical or irregular. Limitation to one eye occurs so seldom that it should not suggest nuclear disease. On the other hand, both eyes may suffer from disease of the nerves (trunks, branches, or endings), but such bilateral disease is rare, and, without other evidence of a nerve lesion, should suggest nuclear mischief.

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* It should be remembered that disturbance of function may cause organic changes or determine disease of the nerve, as well as result from such disease.

† 'Neur. Cent.,' 1888, p. 88.

‡ Nuclear ocular palsy is also termed ophthalmoplegia, a term first used by Brunner in 1850. The nature of the cases was pointed out by M. Graefe in 1856 ('Arch. f. Oph.'), and, in 1868, compared with labio-glossal palsy. Förster correctly localised the lesion in 1878 for external palsies; and internal ophthalmoplegia was well described by Hutchinson in 1878 ('Med.-Chir. Trans.'), and the external ophthalmoplegia, with post-mortem proof of its nature, in 1879 (ibid.).

§ A name open to many objections, both theoretical and practical. Nuclear palsy of all the external muscles has, indeed, been associated with polio-myelitis in a man of forty, but the cord affection was acute, and that of the eyes was chronic (Sachs, 'American Journ. Med. Science,' 1889).

(1) *Chronic nuclear palsy, chronic ophthalmoplegia,* includes the isolated loss of the reflex action of the iris, isolated palsy of the ciliary muscles, palsy of all the internal muscles, of all or many of the external muscles, and lastly, of both the external and internal muscles. It also includes loss of the upward or downward movements of the eyes, ptosis, and the conjugate lateral palsy already described. The combinations met with are extremely numerous and varied.

Later observations have shown that we cannot separate most forms of chronic palsy. They occur under similar conditions; they may be variously combined, and the dependence of one of them on nuclear disease has been conclusively proved, so that it is probably true of most. Each form is met with in association with locomotor ataxy; the simple loss of reflex action is indeed present in the majority of cases of this disease. It has been mentioned that when this loss exists alone the subjects of it have frequently suffered from constitutional syphilis, and the same fact is true of the other forms of ophthalmoplegia.* They sometimes occur, however, without this antecedent, which may or may not develop later. They occasionally affect young persons, and are met with more frequently in males than in females, resembling in this their congener, tabes. It is seldom that any immediate cause can be traced; in one instance the symptoms are said to have come on after a wetting. I have seen the condition as a late and permanent sequel of diphtheria.

Some other features deserve mention. The first muscles to suffer may be those that are associated in action—the superior recti and levators, the two internal recti, or the internal rectus on one side and the external rectus on the other; or it may be quite irregular. The loss of power, at first slight, slowly increases; often a strong effort shows more power than is habitually exerted, and the weakness may be less in the morning than in the evening. The disease slowly extends, until at last, often after several years, only one or two of the muscles of both eyes retain power, and sometimes every muscle is paralysed. The levators, however, seldom become completely paralysed, and may be unaffected. The aspect of the patients is peculiar; when there is partial ptosis, this gives them a sleepy expression, and in total palsy without ptosis a staring look results from the immobility of the eyes, which are fixed in mid-position. Sometimes, although rarely, they are slightly prominent. Occasionally one eye is much more affected than the other; on one side there may be total palsy, on the other only ptosis, or loss of the light-reflex. The internal muscles are often unaffected; when this is the case the diagnosis of nuclear palsy can be made with confidence, because the escape of the internal muscles in bilateral disease of the nerve-trunks is scarcely possible. This feature was thought to be a characteristic of progressive ophthalmoplegia by von Graefe, but it is not so; the affection of the external muscles may be combined with loss of reflex action of the iris, with cycloplegia, or with both as "total ophthalmoplegia." In the case presently to be mentioned, in which the nature of the lesion was ascertained, the internal muscles were affected. Double vision may trouble the patient in the early stage of the affection, but it generally passes away as the disease progresses, and is sometimes absent from the first, perhaps when the early loss is of associated movements. The duration of the malady is long. In one of Hutchinson's cases the symptoms continued for seventeen years. In one of Mauthner's the affection is said to have been limited to one eye for twenty years. It is common for arrest to occur when a certain degree of palsy has been attained. The symptoms may exist alone, but they are frequently associated with indications of other diseases of the nervous system—with optic nerve atrophy, with affection of the bulbar nerves* (rarely amounting to typical bulbar palsy), with general paralysis of the insane, and especially with locomotor ataxy. It may attain a considerable degree before there are any symptoms of tabes, even when this is ultimately severe. A similar condition is perhaps sometimes congenital, and even hereditary.†

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* As in so many of these degenerative diseases, the facts that can be ascertained probably under-represent the relation to syphilis. The case of Mr. Hutchinson, examined by me (see p. 197), is an illustration of this. The man persistently denied any venereal sore, and no trace or history of syphilitic disease could be found. But some years later a child of this man was brought to Mr. Hutchinson with characteristic notched teeth and interstitial keratitis.

The nature of these cases is not a matter of speculation. In a case of Mr. Hutchinson's, in which I examined the brain (1879), the state of the nuclei of the ocular nerves was nearly the same as that of the grey matter of the spinal cord in progressive muscular atrophy. The patient was a man, aged fifty-five, who had had syphilis. The symptoms began gradually, seven years before death, with palsy of the internal recti and ciliary muscles. Ultimately all the ocular muscles became very feeble, the optic nerves atrophied, there was mental excitement, and some palsy developed in the limbs, the condition resembling that of some forms of general paralysis of the insane. The roots of the ocular nerves, outside and inside the brain, were grey, small, and contained scarcely any normal fibres. In their nuclei a few nerve-cells of normal size were seen, but these had, for the most part, lost their processes, and a large number of the cells were reduced to small angular bodies or had disappeared (Fig. 101).

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* This combination existed in the case that was a late sequel of diphtheria.

† Thus Hirschberg ('Berlin. Gesellsch. f. Psych.,' June 8th, 1885) has described the case of a man with congenital double ptosis and paralysis of all the ocular muscles, incomplete in the superior oblique and the internal muscles, whose mother presented a similar condition, while his son had congenital ptosis and paralysis of the superior recti.

In the intervening tissue there were many connective-tissue nuclei. The change involved the sixth, fourth, and the whole of the third nuclei. The other nerve-roots and nuclei were normal. The spinal cord was not obtained. Thus the lesion, in a fairly characteristic case, was a degeneration, limited to structures having a common function. The internal palsies occur under similar conditions, and their frequent limitation to a single function in the two eyes points to a like change of more restricted seat. It is only by toxæmic influences or degeneration that structures of common function are selected, as it were, for isolated disease, and it is probable that all degenerations are often the effect of a toxæmia (see "Tabes," vol. i), perhaps a chemical "toxine," produced by organisms. In other and younger subjects they seem to be due to deficient vital endurance of the structure, varying in degree so as to be manifested at birth or not until adult life.

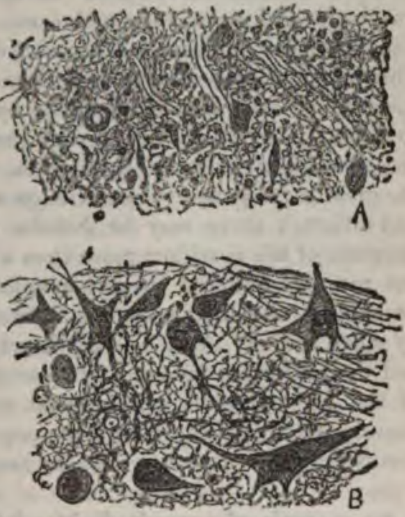

FIG. 101.—A, part of sixth nucleus in progressive ophthalmoplegia; B, cells of the normal nucleus, for comparison.

But in all degenerations destruction begins as slight and slow changes in nutrition, doubtless sometimes capable of recovery, as when internal palsy passes away after it has lasted for many months, and perhaps for more than a year. I have known the light-reflex to return, in stationary tabes, after it had been lost for at least two years, and the remarkable case of internal ophthalmoplegia recorded by Eales (see p. 185) is an instance of the way in which such a palsy, limited and complete, may pass away. Such recovery is especially intelligible if the changes are due to a toxæmia.

Infantile oculo-facial palsy is a rare variety of the chronic form, which may be congenital, or begin at some period of childhood, rarely in early adult life. Palsy of the external ocular muscles, all or many, is very commonly accompanied by paralysis of the face, sometimes chiefly of the zygomatics. Ptosis is common, but the internal muscles do not suffer. It usually becomes stationary, and remains so through life. Nothing definite is known of its causation, but its history suggests defective vital endurance in the nuclear structures.*

Sudden Nuclear Palsy.—The vascular lesion which causes sudden palsy, and the onset of which is survived, is usually obstruction of the arterial branches which the basilar gives to this part. The obstruction is usually bilateral, because disease in the wall of the basilar, whether atheroma or syphilitic, affects the branches given off at the spot on each side. Hence small foci of softening are often found in each third nucleus. The same conditions determine the bilateral character of the lesion and symptoms in the rare cases of embolic obstruction. But the lesions are irregular in distribution, and the symptoms unsymmetrical as a rule, and even partial, different parts of the nuclei being supplied by different branches, according to Heubner. This irregularity is a difference from the chronic form, while other distinctions are furnished not only by the onset, but by the tendency to improvement which they, and all similar lesions, present, and by the occasional association of hemiplegia, usually on the side opposite to the greater eye-palsy. Hæmorrhage, when it occurs, is apt to spread, and although there may be definite ocular palsy at the onset, the duration of life is seldom more than a few hours. Only in rare cases has recovery followed irritant apoplexy, that has made hæmorrhage probable.* An instructive example of the embolic form is recorded by Allen Starr.† A man with heart disease had sudden giddiness and double vision, followed by somnolence, unequal pupils, paralysis of the right inferior and internal recti, and the left inferior and superior recti. In eight days the pupils were equal, and the internal rectus normal; the other muscles remained paralysed at the end of a year.

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* Möbius (1892) has collected forty-four cases ('Munch. med. Wochenschr.').

An example of a much slighter lesion, probably due to syphilitic vascular disease, and characteristic in its course and irregularity, was that of a man who, one day, suddenly found both upper lids drooping, and then that both eyes deviated outwards, and could not be moved. In a few days he could move the right eye fairly well, but six months later he presented complete internal ophthalmoplegia in both eyes, with, in the left, slight ptosis and complete palsy of the other muscles supplied by the third nerve, but not of the external rectus, and the superior oblique possessed a little power.

Acute and subacute nuclear palsy includes the cases which reach their height in a few days or a few weeks, an onset that suggests one of two morbid processes—inflammation, or the action on the centre of some poison circulating in the blood, to which, indeed, actual inflammation may sometimes be due. But such a poison may abolish function without causing visible changes; hence the name proposed by Wernicke‡ and widely used, "polio-encephalitis superior," is not always correct. Even the less objectionable "acute nuclear palsy" may prove to be sometimes inaccurate, since the symptoms may be due to peripheral neuritis and not to nuclear disease. Hitherto, however, most post-mortem examinations have shown evidence of inflammation in the nuclei beneath the corpora quadrigemina, softening, minute extravasations, &c. Thus in two cases of external palsy, complete except that the levator escaped, Thomsen found great hyperæmia of the nuclei, third, fourth, and sixth (and of the hypoglossal), with degenerative changes in the nerve-elements. But the malady is rare, pathological observations are very few, and our knowledge of the cause of this form is therefore meagre. Of three cases described by Wernicke, one followed poisoning by sulphuric acid, and the other two in the subjects of chronic alcoholism. Injury has been supposed to be an occasional cause.

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* See Sturge, 'Trans. Oph. Soc.,' vol. i.

† 'Journ. Nerv. and Mental Dis.,' 1888.

‡ Who first accurately distinguished this form ('Gehirnkrankheiten,' ii, 233). It was also well described by Thomsen ('Arch. f. Psych.,' 1888). Good descriptions have been given by Mauthner ('Vorträge').

Eye-symptoms usually exist alone, the combination with bulbar palsy being seldom met with except in the chronic form. In a few cases, however, acute imperfect bulbar palsy has spread to the upper part of the face, and then to the eye muscles, a form that has been termed "ascending inferior polio-encephalitis."

In the usual form of acute nuclear palsy the eye muscles suffer in the same irregular manner as in the sudden variety. It is common for the internal muscles to escape: the levator may or may not be involved. In fatal cases the motor influence spreads, becomes extensive, both in the centres of other nerves and in the cortex, and the cerebral functions suffer so widely as to cause death in one or two weeks. In the cases that survive there is some improvement in the ocular palsy, which may be considerable, so that there is only slight residual impairment, or considerable palsy is limited to one eye. This is explained by the random character of inflammatory damage.

In the rare cases in which a lesion is limited to the grey matter of the corpora quadrigemina, the chief eye symptom appears to be a loss of certain conjugate movements, especially of that upwards. But acute lesions usually produce other effects which mask those in the ocular muscles.

Peripheral Ophthalmoplegia.—Palsy of the muscles of both eyes, resembling the nuclear form, may be due to peripheral neuritis. How relatively frequent this form is we cannot say, since the chief cause of peripheral neuritis, alcoholism, may also cause inflammation of the nuclei. Neuritis may be suspected when many other nerves suffer at the same time, or alcoholic multiple neuritis co-exists in the limbs,* although the latter cannot be regarded as proof, since alcohol may affect the nuclei. A case of neuritic ophthalmoplegia, which may be regarded as typical, is that of a man of sixty-two in whom all the eye muscles became weak as well as the levator palpebræ. The fifth nerve suffered, swallowing became difficult, and death followed. The ocular nuclei were normal, but there was wide-spread peripheral neuritis of both cranial and spinal nerves.†

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* Suckling, 'Brit. Med. Journ.,' 1888.

† Meyer, 'Neur. Cent.,' 1888.

OTHER FORMS OF PTOSIS.—*Reflex Ptosis.*—In very rare cases irritation of the fifth nerve has been observed to cause transient drooping of the eyelid, which must be referred to inhibition of the centre for the muscle. A physiological connection doubtless underlies this effect; spasmodic closure of the lids frequently results from irritation of the fifth nerve; for closure of the lids there is always relaxation of the levator (as is shown by the slight amount of orbicular contraction which will suffice), and hence irritation of the fifth nerve has a tendency to cause relaxation of the levator. Reflex ptosis has been observed to follow division of the fifth nerve, no doubt from the irritation of the fibres by the section (Longet). It may result from the extraction of a tooth, as the following case shows. An upper right molar, decayed and causing much pain, was extracted from a woman aged fifty-five. The tooth was firm, and the patient had no anæsthetic. A few hours afterwards right-sided ptosis existed, varied by occasional attacks of clonic spasm in the levator, each lasting a few seconds. Both symptoms continued, in slighter degree, on the following day, and gradually improved, so that the condition of the eyelid by the fifth day was natural. On the sixth day there was some pain referred to all the branches of the fifth nerve, but this soon passed off, and there was no recurrence of nervous symptoms.*

Ptosis from Paralysis of the Sympathetic.—The unstriated muscular fibres (fibres of Müller), which exist in the fascia of the orbit, and are innervated from the sympathetic, act indirectly on the tarsal cartilages by the connection of these with the fascia. They probably aid to a slight extent in maintaining the upper lid in its normal position. When the cervical sympathetic is paralysed, the upper lid on that side is a little lower than on the other. Its movements are unimpaired. Ptosis from this cause is distinguished by the presence of other symptoms of paralysis of the sympathetic, such as contraction of the pupil, and sometimes dilatation of the vessels of the surface or altered secretion of sweat.

Congenital ptosis, not due to malformation of the lid, is usually bilateral and partial. It is generally associated with defective power of elevation of the globes, and is probably due to a congenital central defect. In one case there was also a defect in the outward movement of the eye, associated with an inward movement of the other, but absent when the eye was moved alone. Congenital ptosis may be hereditary, and is probably allied to the more extensive infantile nuclear palsy already described. Slight double ptosis sometimes runs in families, and may (as I have seen) affect chiefly the female members, sometimes coming on after puberty, and occasionally after middle life. The characteristic over-action of the frontal muscles causes the anxious aspect already mentioned, and this may actually be more conspicuous than the slight drooping of the lids to which it is secondary.

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* Communicated to me by Mr. H. R. Gooding, by whom the case was observed.

Allied to this is a form which may be termed *morning ptosis.* During sleep the levator is relaxed, to permit closure of the eyes by a gentle contraction of the palpebral orbicularis. Many sound sleepers find a difficulty in opening the eyes on being first roused. In weakly women this difficulty is sometimes increased; after waking, it may be impossible for them to raise the eyelids for ten, fifteen, or thirty minutes. During the rest of the day they have no difficulty. This condition is usually recovered from.

Hysterical ptosis is occasionally met with, single or double. It is generally accompanied by a slight spasm in the orbicularis; this can readily be proved by making the patient look upwards, when the spasm of the orbicularis becomes much greater to prevent the lid from moving with the eyeball. When double* (Fig. 102) both eyelids droop, and the patient puts her head back when she is told to look up. If the head is held in the attempt to look up, both orbiculares contract, and prevent the lids rising. This contraction of the orbiculares proves that there is no true paralysis of the levators. There is sometimes over-action of the frontales associated with the ptosis, as if from a struggle to overcome the contraction of the orbiculares.

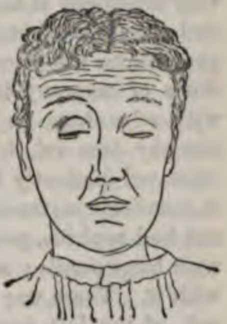

FIG. 102.—Hysterical ptosis, bilateral. Over-action of frontales.

TREATMENT.—The most important element in the treatment of paralysis of the ocular muscles is that of the morbid process that causes them, and this is described elsewhere. It is only necessary here to mention those measures that the special effect of the disease renders necessary. Whenever the onset is acute or subacute, or if other symptoms suggest inflammation, counter-irritation should be employed. A blister may be placed behind the ear or at the occiput when the disease is probably at the base of the brain, on the temple if it is in the orbit. The blister is often followed by a striking increase in the power of the affected muscle. In acute rheumatic cases hot fomentations should be applied to the orbit, forehead, and temple, frequently repeated. Leeches may be applied to the temple, at the onset if the patient's general condition does not contra-indicate their use. In syphilitic cases appropriate remedies should be freely employed. In most cases of local neuritis, indeed, whether syphilitic or not, mercury is probably the most efficient agent we possess. Defective general health must be corrected by tonics, of which iron and strychnia are the most useful.

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* A case is recorded by Debove ('La Semaine médicale,' 1890) of paralysis of both third nerves with double divergent strabismus, said to be hysterical. The condition came on after an accident.

For nuclear palsy, in syphilitic subjects, mercury and iodide have seemed useful only when the onset was acute or subacute. It is probable that in some instances these drugs have cured the patient, but in other cases it is doubtful whether the improvement has been more than the tendency to recovery which usually follows an acute process that has spent its force. In cases that are chronic from the beginning this treatment is as powerless as it commonly is in locomotor ataxy, and the most effective measures are those suited to primary nerve degenerations elsewhere—arsenic, quinine, strychnine, and small tonic doses of mercury in syphilitic patients. Strychnine may be given with advantage by hypodermic injection, as in progressive muscular atrophy (see vol. i, p. 497). Such treatment as is here suggested appeared distinctly beneficial in one case, the sequel of which renders it peculiarly instructive. A gentleman aged forty, who had probably not had syphilis, presented entire loss of the reflex action of each iris, with large pupils; accommodation was normal, and the iris contracted with it. There was also slight double ptosis, weakness of both internal, of the left superior, and of the right inferior recti, and great diminution in the right knee-jerk, all of gradual onset and from six to eighteen months' duration. At the end of a three months' treatment the external ocular muscles had become normal, although the reflex action was still lost, and the right knee-jerk had increased to the same degree as the left. He continued without relapse for nearly two years, and then suddenly presented the acute mental derangement of general paralysis of the insane, from which he quickly died. But we do not yet possess the means of combating with success the toxæmic influences that are probably the most frequent cause.

Recorded cases of the recurrent or periodical palsy do not show that any treatment has a marked influence on the attacks. Early counter-irritation probably affords most prospect of modifying their course, and tonic treatment may be adopted before an expected attack. In cases in which the attacks recur frequently, the treatment adopted should be that for migraine.

Direct Treatment.—Electricity has been recommended and employed in the treatment of these paralyses, but its influence is not great. Direct application to the affected muscle is scarcely practicable. It has, indeed, been carried out by a very small electrode, or by a wet camel-hair brush in the centre of which is a conducting wire. The extreme sensitiveness of the conjunctiva renders the application most painful, and although the pain may be lessened by cocain, the application of a voltaic current, sufficiently strong to stimulate the muscle, in such proximity to the retina and on such a delicate structure as the conjunctiva, is scarcely safe. In most ocular palsies the muscles do not respond to faradisation. The voltaic current may be applied through the eyelid, in the case of a superior or inferior muscle, the eye being turned in the opposite direction. The considerable diffusive power of this current makes it probable that the electricity would reach the muscle, although even thus a current cannot be safely used of sufficient strength to produce evidence of stimulation. The application of a faradic current through the eyelid is useless, even if the muscle would respond to it, because a thin layer of contractile muscle prevents any stimulation of the subjacent tissues. For these reasons, most authorities are agreed in deprecating any direct application. A method of indirect electrisation has been recommended (by Benedikt and others) which consists in placing one pole (anode) on the forehead, the other (kathode) on the margin of the orbit near the affected muscle. If the faradic current is employed, the orbital pole is kept still; if the voltaic current is used, this pole is stroked along the skin, or the circuit is alternately made and broken by an interrupter. A slight increase of power in the muscle may be observed after the current has been so applied for a few minutes, but it quickly passes off, and in cases free from sources of fallacy I have never been able to trace any permanent effect. It is true that works on electro-therapeutics contain cases in which improvement or cure was ascribed to this agent, but in most instances the authors have ignored the tendency to spontaneous improvement in recent cases, and the influence of drugs, as iodide of potassium, which were given at the same time in many instances.

The diplopia due to partial paralysis of a muscle may be removed by the use of a prism. One strong enough to completely fuse the images is undesirable, because it tends to prevent the strengthening influence of effort. A weak prism, strong enough so to approximate the images as to permit their fusion by muscular action, sometimes does good. It may be used for an hour daily as a gymnastic exercise. The giddiness due to erroneous projection can only be removed by putting the eye out of action by an opaque glass; this glass may be in a pair of spectacles, the other glass being merely tinted; the opacity is then inconspicuous to an observer. But this has the same disadvantage of withdrawing the stimulus of exertion. An opaque glass over the sound eye is useless for the vertigo, and increases the amount of the secondary deviation.

Operative interference can do little in cases of ocular palsy. The only condition in which it is admissible is that in which antagonistic contracture has been developed, and the paralysed muscle has regained power, but cannot overcome the opposing contracture. In such a case the tendon of the contractured muscle may be divided without disturbing its other connections; it forms a fresh attachment a few millimetres further back, and the result on the movement of the eye is often very satisfactory.

The treatment of paralytic *ptosis* is that of disease of the third nerve. The muscle itself is not accessible to electrical stimulation. In the double ptosis of nervous debility, nervine tonics, quinine and strychnia, are useful, and their influence is aided by local treatment, which stimulates the fifth nerve, and so produces a reflex action on the centre of the levator. Electricity may be used, either the voltaic or induced current applied to the skin about the orbit, but a stimulating liniment which stings the skin (as chloroform liniment on spongiopiline) usually answers as well. The morning ptosis can always be quickly relieved in this manner. The hysterical form is often a very obstinate affection. Blisters to the temple, faradism, and (in the unilateral form) tying up the other eye, are the most efficient means, and usually in time remove the symptoms.

SPASM OF THE OCULAR MUSCLES.

The varieties of spasm of the ocular muscles are numerous, but only a few are of medical significance. Two classes may be dismissed with a mere mention. (1) Those that are connected with disorder of the ocular visual process, as the convergent strabismus of hypermetropia, the divergence of myopia, the adaptive deviation that occurs when there is partial opacity of the media, and the irregular position that often accompanies absence of sight. (2) The secondary deviation in one eye, consequent on palsy of a muscle of the other eye, and the antagonistic contracture in the same eye. The second class has been already described; those of the first do not come within the scope of this work. Of the remaining forms, one of great importance—nystagmus—will be separately described. The remainder may be grouped into five classes.

(1) *Associated spasm from central disease* is not uncommon. Its character depends on the position of its cause: both eyes are affected in disease of the hemispheres; one eye only in some cases where the lesion is in the nuclei or the eye muscles. In a paralysing lesion of one hemisphere the eyes deviate towards the diseased side, but the deviation is merely due to the unopposed influence of the opposite hemisphere. An irritating lesion of one hemisphere causes conjugate deviation towards the opposite side, the result of spasm; this occurs also at the onset of unilateral convulsions, and is often associated with muscular rigidity of the limbs on the side towards which the eyes are directed. In one interesting case, deviation to the right was, during four months, a persistent symptom of a depressed fracture below the left parietal eminence, and it ceased when the depressed bone was raised by trephining.*

Since a lesion in one side of the pons causes a loss of the conjugate movement towards the side of the lesion, the muscles being excited from structures in this position (see p. 186), irritating disease may be expected to cause spasmodic deviation towards that side. But such spasm is seldom observed, apparently because the structures concerned are readily damaged and rendered inactive. An acute lesion which causes the conjugate paralysis may, however, produce a distinctly spasmodic deviation towards the opposite side, probably by an indirect influence on the corresponding centre of the other side of the pons.[*] Between the two there must be a close functional relation, indispensable for their normal action, and throwing light on many phenomena of disease (see "Nystagmus," p. 209).

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* Thompson, 'Brain,' April, 1883, p. 99. It may be noted that the fracture was behind the region in which experiment places the centres for the lateral movement, which is in front of the central convolutions (see p. 13). But the structures that overact are often only near a primary lesion, the influence of which is exerted on the neighbourhood, and sometimes only in one direction.

(2) *Irregular spasm from brain disease.*—In irritating diseases of the base of the brain, especially in meningitis, spasm occurs in one or more of the ocular muscles, causing slight irregular deviation of the visual axes, comparable to the variable rigidity that occurs in the limbs, and may change in seat and degree from time to time. It is probably due to irritation of the motor nerve-trunks. Care must be taken not to mistake for spasm the deviation from loss of power which is so common from the same cause. In spasm there is deviation when the eyes are at rest in the mid-position; in recent paralysis there is not. It must be remembered that paralysis and spasm are often conjoined. Such irregular spasm is generally tonic.

Slight irregular spasm of the ocular muscles sometimes occurs in chorea. It is rarely sufficient to be seen, but causes transient diplopia, which may be thought to indicate organic brain disease if the dependence on chorea is not known. If patients are questioned on the point, this diplopia will be found to be by no means rare.

(3) *Chronic spasm in individual muscles* is extremely rare apart from the secondary deviation already mentioned. In the best-marked cases the spasm has not been continuous, but has occurred on certain movements of the eyes, sometimes associated with pain. Thus, of two cases recorded by Hock,[†] in one, as soon as an object was moved to the right of the middle line, spasmodic contraction occurred in the right internal rectus (with great pain), bringing the eye into extreme adduction, and as soon as the object was moved to the left beyond the middle line the spasm relaxed, and the right eye moved outwards into the fixing position. In the other case there was slight weakness of the right internal rectus, and as soon as the object was moved to the left of the middle line, spasm in the left external rectus moved the eye into extreme abduction. The spasm thus affected the muscle that would be the seat of secondary deviation, but was distinguished from this by its extreme degree. This case is interesting because the spasm occurred with slight left hemiplegia, and, from some accompanying disturbance of taste, may have been due to disease in

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[*] As in a case I have recorded of acute anæmia of the right half of the pons (due to arterial obstruction) which caused deviation of the eyes to the left. It was increased from time to time, with violent nystagmus ('Trans. Ophth. Soc.,' 1884, p. 208).

[†] 'Wiener Klinik,' April, 1876, p. 116.

the upper part of the pons. Sometimes two muscles acting together are involved in the spasm, as, in one case, the superior oblique and inferior rectus, which are normally associated (Stilling).

(4) *Hysterical Spasm.*—In hysterical fits the eyes are usually directed either upwards and to one side, often so as to almost entirely conceal the cornea, or they are directed inwards in strong convergence—a common symptom. They never diverge as they do in organic disease. Sometimes the convergence persists during the intervals, and is then usually associated with spasm of accommodation.

(5) *Paroxysmal Spasm.*—In convulsive attacks in which the convulsion is unequal on the two sides, the eyes constantly deviate (with the head) towards the side most convulsed, and if the second side is affected in greater degree after the first, the eyes subsequently deviate towards that side. When the fit is over there may also be a deviation from the side most convulsed, accompanying the transient weakness common in that side. Such spasm is seldom unilateral, but during brief attacks of clonic torticollis (in a child of eleven months) drawing the head to the right, there was strong deviation of the left eye in the same direction. A single muscle may be the seat of brief spasm, resembling in miniature an epileptoid seizure, and sometimes attended with transient obscuration of consciousness. During the attack there is diplopia, and often giddiness from the erroneous projection. Clonic spasm in the orbicularis may occur at the same time. Such attacks, for instance, occurred in a man aged forty-seven ; there was very slight permanent defect of power in the left external and both internal recti. Occasionally, without any exciting movement of the eye, tonic spasm in the external rectus would draw the left eye strongly outwards for about thirty seconds. During this time there was constant winking with both eyelids, which seemed to the patient to be due to an effort to get the eye right, but could not be prevented. The deviation suddenly ceased, but for a few minutes afterwards the left eyelid was about one twelfth of an inch lower than the other, and then both were alike. There was no history of venereal disease, and syphilis was for other reasons very improbable.

Another patient, aged thirty-six, also without history of syphilis, suffered from frequent attacks (lasting only a few seconds), two of which I witnessed. There was a sudden sensation of heat, spreading from the left inner canthus and extending over the eye and temple, accompanied with impairment of sight of both eyes, varying from slight dimness to absolute loss of sight, and with abduction of the left eye from mid-position to about halfway to the outer canthus, the right eye being still. If walking, he deviated to the left during the attack, probably from erroneous projection of the left field. In the intervals, movements of the eyes were perfectly normal. The attacks consisted of tonic and then clonic spasm, and resembled perfectly miniature epileptic convulsions. Some years later this spasm had ceased, but the patient presented complete internal and partial external ophthalmoplegia, clearly due to nuclear degeneration.

NYSTAGMUS.—Rhythmical movements of the eyes, involuntary, frequent, usually bilateral and similar in each eye, produced by alternating contractions in opposing muscles, are termed "nystagmus." Two forms must be distinguished: (*a*) that in which slight movement is associated with weakness of the muscles, and occurs only when the weak muscles are put in action; (*b*) that in which the movement goes on continuously in all positions or only in some, with or without loss of power, but more than the mere tremor of weakness. Only the second form is here considered. Among its causes the following are the chief:

(1) Local affections of the eyes which interfere with sight, but have no other character in common,—opacities of cornea or lens, and inflammations or degenerations of retina and choroid. It scarcely ever results from simple errors of refraction, however considerable. These eye diseases cause nystagmus chiefly when they occur in infancy or early childhood. Nevertheless it is doubtful whether nystagmus results from blindness which is actually congenital, although slow rolling movements of the eyes are observed in such cases. In adult life ocular disease alone rarely causes nystagmus, but it certainly aids the development of nystagmus due to other causes.

(2) In albinism this condition is very common.

(3) It occurs in miners, chiefly in those who work in coal-mines, and who use the pick in a stooping or lying posture, in which the eyes are habitually turned to one side. It is also far more frequent where the dim safety-lamp is used than in mines which can be worked with brighter naked lights. This form has been admirably investigated and elucidated by Snell.

(4) It occurs in diseases of the nervous system of the most varied seat and character, especially in degenerative affections of most forms, but sometimes accompanies the chronic stage of acute maladies, such as disseminated myelitis. It is usually conspicuous in insular sclerosis and also in hereditary ataxy, but not in ordinary locomotor ataxy. In other diseases attended with tremor it is rare. In paralysis agitans it is never met with—a curious fact, considering how closely the alternating movement of nystagmus resembles that of shaking palsy. It occurs in many diseases of the brain, diffuse and focal; meningitis, meningeal hæmorrhage, thromboses in sinuses, and in cases of tumour, hæmorrhage, and softening in various situations. It is common in tumours of the cerebellum, but the most intense nystagmus I have seen was due to a tumour in the right optic thalamus. I have also known it to occur in disease of one side of the pons (arterial occlusion causing local anæmia, fatal before softening could occur); the quick movement was from the side of the lesion, and there was associated palsy of the lateral movement towards the

diseased side. In an analogous case, seen only during life, a chronic lesion, evidently on the right side of the pons, caused partial palsy of the right fifth nerve,* and loss of the movement of both eyes to the right. An attempted movement to the right produced no nystagmus, but when the eyes were moved to the left this at once set in, the quick motion being in that direction. Nystagmus often occurs in cases of degenerative disease of the spinal cord and brain, in which there is no evidence of a focal lesion.

The movement is usually bilateral; very rarely it affects one eye only. Horizontal (lateral) movement is the most common; next a rotatory motion; vertical movement when the eyes are directed upwards will be found far from rare, if looked for, but is seldom continuous when the eyes are at rest; one-sided nystagmus has been said to be generally vertical. Rotatory nystagmus may occur only when the eyes are moved in a certain direction. Its character may show clearly its sources; *e. g.* in one case the quick movements were manifestly due to the superior oblique. I have once seen the motion limited to the internal recti and the mid-position, ceasing, or almost ceasing, on lateral movements. It was a case of tumour of the right optic thalamus, which might have pressed on the corpora quadrigemina. It probably did so, since the upward movement and right external rectus were enfeebled. The extent of the movement in nystagmus varies from one to ten millimetres; from two to four millimetres is the common range. Occasionally, when it is too slight to be seen with the naked eye, it may be observed with the ophthalmoscope when the fundus is examined. The frequency is usually from 60 to 200 separate movements (*i. e.* in the same direction) per minute; rarely it is slower than 60; occasionally it is too frequent to be counted. The rhythm is usually regular; in some cases there are slight variations in frequency from time to time. If there is slow nystagmus when the eyes are at rest, the oscillations often become more frequent when the eyes are moved. The alternating movements are not equally quick; there is a sudden rapid movement in one direction and slow return. In describing nystagmus, it is said to be to the side towards which the movement is most rapid. In many continuous cases the quick movement is in the direction in which the nystagmus is most energetic on voluntary movement.

The symptom may be constant, or it may occur only when the eyes are moved in a certain direction, sideways, upwards, or downwards. In the second form it is often associated with weakness of the muscles, to which it may be due. Very rarely there is a slight movement of the head, corresponding to that of the eyes in time and in direction, or in the opposite direction to that of the eyes.† All forms cease during sleep. In cases that date from infancy the patient is never conscious of the movement, nor is there any apparent movement of objects; in cases which commence in later life there sometimes is an apparent movement of objects; more frequently there is not. The apparent movement of objects is generally in the direction of the quick movement of the nystagmus. Rarely there is a movement of the upper lid synchronous with that of the eye; this association occurs chiefly when the nystagmus is vertical, but I have seen it once when the movement was horizontal. In vertical nystagmus there is often a slight movement of the lid communicated to it from the eye, which must be carefully distinguished from the actual spasm of the lid.

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* Chiefly of the motor division, accompanied by slight paralysis of all parts of the right side of the face, constant blinking of left eyelids, not of the right, and vertigo, objective and subjective, to the left.

† In a case lately under my care, with symptoms of cerebellar tumour and lateral

The nystagmus of miners presents considerable variations in different cases. Any form of movement may be met with, and it may occur in all or only in one position of the eyes. It often occurs only in the recumbent posture, such as the miner assumes in his work, and may cease when he is upright.*

The physiological pathology of nystagmus is still to a large extent obscure. The perfect bilateral symmetry of the movement, conspicuous in the vast majority of cases, indicates its central origin, and is opposed to the simple but inadequate explanation which ascribes it to muscular fatigue. Why the steady tonic contraction of health should be broken into clonic contraction no one has yet been able to say; but the physiological associations of the ocular movements enable us to understand something of the influences that produce it. We may take as the simplest form that in which the movement is lateral. For lateral movement of the eyes, there is a separate centre in the pons on the side towards which the movement takes place. The movement of lateral nystagmus, like that of health, must be directly produced from this centre. Every normal movement involves the activity of the centre, and also a lessened action of that on the other side, causing relaxation of the antagonists, and this is followed by increased activity, so as to bring the eyes back to the middle line.† Hence there must be a mutual connection between the functional states of the antagonistic centres on the two sides, and this probably underlies the alternation of movement in nystagmus. It is probable that the intermittence is due to a derangement of their function, necessarily produced whenever the eyeballs are the seat of spasm. If so, the oscillations may be neglected in considering the

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nystagmus, the pharynx and larynx were the seat of similar movement; that in the pharynx was horizontal, towards the middle line; in the larynx there was a similar lateral movement of the arytænoid cartilages. The rate of the movement was the same as in the ocular muscles, 180 per minute (Spencer, 'Lancet,' 1886, vol. ii, p. 702).

* See Snell, 'Trans. Oph. Soc.,' vol. iv, p. 315.

† That mere elasticity brings the eyes back seems improbable from what we know of the related contraction of antagonists elsewhere. Moreover any such action depends on muscular tone, and is absent in the atonic palsy of total nerve disease.

relations of the symptom. Their origin constitutes a separate problem common to all forms, and distinct from that of its causation.

The relations of nystagmus are extensive. The nuclear centres are acted on by centripetal impressions as well as by the will. They are influenced by visual impressions to a degree which we can only discern indirectly (since the process does not enter into the region of consciousness) by considering how accurately and yet how easily we can follow with the eye a swiftly moving object. This ability is apparently acquired. The newly born child never "fixes" an object, never follows a light, however brilliant. Not until later is there established the functional relation of the motor centres to the visual impression, in what is essentially a reflex process,* and for this to be normally developed it is apparently necessary that the stimulation of the optic nerve should correspond to the average degree and character. Hence it is intelligible that if this visual control is partially but not perfectly established (in consequence of early ocular disease lessening vision) the action of the motor centres should be abnormal. Hence, too, we can understand that imperfect ocular guidance (as the bad light of the safety-lamp) may aid other causes in developing derangement in later life. In albinism the early visual impressions may err on the side of painful and disturbing excess, from the absence of the pigment of the eye.

Again, the ocular motor centres must be acted on by, as they certainly act on, the equilibrial centres. These are guided by the innervation of the eyeball muscles. Thus these muscles will be brought indirectly under the influence of the semicircular canals. Nystagmus can be induced in health by rotation of the body (Donders); quick movements of the eyes occur in the direction in which the body has rotated, and there is a slower return. The quick movements occur towards the side on which the horizontal semicircular canal has been subjected to increased pressure by the rotation. In chronic otitis, pressure on the ear has been often observed to cause a nystagmus precisely similar, ceasing with the pressure; † it is usually lateral, but sometimes rotatory. Vertigo always attends it; a motor sensation which, if intense, goes on to movement in which the eyes and head lead the way. Hence involuntary movement of the eyes is not surprising, the cause of the intermittence being, as already stated, a separate problem. In the nystagmus of miners, which may occur only in the recumbent posture, we may also trace, as one factor, an influence of the equilibrial centre (and probably of the canal impressions) on the centre for the movement of the eyes.

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* I recorded some years ago ('Brain,' vol. ii) a case in which the reflex fixation of the eyes was brought into salience by disease. If the patient, looking at one object, was told to look at another at some lateral distance from the first, his head was instantly turned in the direction of the second object, but the eyes remained fixed on the first by a movement as rapid as that of the head, but in the opposite direction, and then they were slowly moved into the position corresponding to the second object. The patient was in the last stage of progressive muscular atrophy.

† Schwalbach, Hughlings Jackson, Pflüger (attempt to extract a polypus). Injections into the ear sometimes excite it. Movements are also related to true auditory impressions, at least in so far as rhythm is concerned, as musical "marches" illustrate. In peculiar hysteroid states, Hogyës has found that nystagmus could be produced by bringing a vibrating tuning-fork near the ear, and that the movements varied with the rapidity of the vibrations. He was also able to cause nystagmus by other sensory impressions ('Orvosi-Hetilap,' 1886, and 'Cent. f. Nervenh.,' p. 526).

The wide variation in the position of organic disease that may cause nystagmus is scarcely surprising when we consider how wide and various must be the total connections of the functions of vision, of movement of the eyes, and of the maintenance of equilibrium; and that we can trace a connection between nystagmus and the derangement of each of these, including some causes of vertigo. But it is also common in various degenerative diseases in which we cannot trace its source, but which show how easily deranged must be the mechanism by which in health the muscular contractions are kept uniform. It would even seem as if the nuclear centres have a tendency to rhythmical or intermittent action, which is normally counteracted, possibly by the influence of the corresponding centres on the other side. When deranged, spasm that would be continuous in the limbs becomes intermittent in the eyes. This view is suggested by the case of arterial obstruction in the pons already mentioned. Nystagmus was clearly due to the over-action of the centres on the undamaged side, under the influence of loss of function of those on the other.* But this part of the subject needs far more systematic study than it has yet received.

The practical significance of nystagmus in diagnosis is extremely great, not from any distinct indication of the seat or precise nature of the disease, but because it shows the presence of more than merely functional disturbance. It is often marked at the early stage of degenerative disease when other symptoms are equivocal, and a search for it should never be omitted, and should always include the upward movement of the eyes. It may be trusted without hesitation, and prevents a mistake in diagnosis in a very large number of cases.

Spasm of the levator is occasionally met with as an isolated symptom, and also as one of the features of exophthalmic goitre. The isolated form (sometimes termed *lagophthalmos*, or "hare-eye") is usually due to some inhibitory irritation in the region of the fifth nerve. Late in life it may come on as an independent affection, analogous to other muscular spasms, as torticollis and facial spasm. The contraction is usually tonic, an excessive degree of the normal tonic contraction of the levator which keeps the eyelid raised; the muscle is imperfectly relaxed when the eye is directed downwards or the lids are closed. Hence the eyelid is a little higher than the other when the eyes are directed straight forwards; and on looking down, the lid does not descend, so that a wide extent of sclerotic is exposed above the cornea. When the lids are closed, the upper lid may be brought down by the orbicularis, but not to the same extent as on the other side, and hence the lids do not descend equally. The exposure of the sclerotic above the cornea gives the impression of slight prominence of the ball. A long-continued spasm in the levator seems, indeed, to cause the eyeball to be actually a little more prominent than the other, in consequence of the origin of the levator being below the level of the upper part of the eyeball; but the apparent prominence due to the exposure of the sclerotic is much greater than the real prominence. The retraction of the upper lid may cause a slightly greater fulness of its tissues than exists on the other side. Unless the affection is due to removable irritation of the fifth nerve it is extremely obstinate.

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* In continuous contraction the muscular fibres contract alternately; in clonus all seem to contract together, and the contraction is brief but renewed. Such a common brief contraction would explain the intermissions of nystagmus, the opponents being always ready to contract when the original action ceases. This readiness prevents the assumption that the common action of all the fibres is due to any other cause than the condition of the centre of the acting muscles.

Clonic spasm is also rare. It occurred from time to time in the case of reflex ptosis mentioned on p. 200. A very singular case of congenital spasm of the levator was exhibited at the Ophthalmological Society by Mr. Marcus Gunn. There was slight ptosis and slight myosis on one side, and the eyelid was raised by slight contraction of the levator whenever the external pterygoid of the same side was put in action.*

Spasmodic elevation of the upper eyelid also occurs from irritation of the cervical sympathetic. We have seen that this nerve supplies the plain muscular fibres of the orbit, which are indirectly connected with the eyelids; and that, as their paralysis may cause slight drooping of the lid, so their stimulation may produce slight elevation. It is probable that this spasm is the cause of the increase in the normal elevation, and of the defective descent of the lid in looking down, in many cases of exophthalmic goitre (Graves' disease). But this form will be considered in the account of that disease. I have once seen a similar slight retraction and defective descent, on the left side only, in a girl of seventeen, who had distinct myxœdematous swelling of the breasts, neck, and face, bilateral, but not elsewhere, and with no symptom of Graves' disease, and also, as a transient symptom on one side only (after anxiety), associated with cardio-thyroid disturbance so slight that alone it would scarcely have been noticed. Similar spasm is said to have been met with in pregnancy as a reflex symptom.

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* 'Ophthalmological Transactions,' vol. iii, p. 283. As one of a committee appointed to report on the case, I had an opportunity of carefully examining it. The simplest explanation for the condition seemed to be that some of the levator fibres of the third nerve arose from the motor nucleus of the fifth nerve. The small size of the pupil might be due to the influence of those cells of the third nerve-nucleus which ought to have been, but were not, connected with the levator.

Treatment.—Little can be done for the treatment of these ocular spasms beyond the removal of their cause as far as this can be effected. Hock's second case was apparently cured by specific remedies, his first case by tenotomy. Hysterical convergence can generally be removed by a small blister to each temple, if the potent influence of neglect is ineffective. The paroxysmal epileptoid form of ocular spasm is very obstinate. Bromides have little influence upon it, and tonics do more good than sedatives. Counter-irritation to the temple sometimes seems to produce a beneficial effect.

In the treatment of nystagmus, the improvement of vision, if this is defective, and the removal of any cause that can be discovered, are the chief measures. The form that occurs in miners needs, as a rule, a change of occupation, or at least of habitual position, so as to relieve the eyes from this postural strain, together with an improvement in the light by which the work is done. For other forms very little can be effected. The application of a feeble voltaic current, from the mastoid process to the closed eyelids, was recommended by Svetlin, but has failed in the hands of others. But the symptom, in many cases, scarcely calls for treatment.

Spasm of the levator is a most intractable affection, unless there is irritation of the fifth nerve or any other cause which can be removed. When no cause can be traced, the most varied treatment by counter-irritation, sedatives, and electricity usually fails. Associated with any symptoms of exophthalmic goitre, the treatment is that of the latter disease.

THE FIFTH NERVE.

The fifth nerve, it will be remembered, has an extensive deep origin, not only from the middle nucleus at the level of its surface attachment, but also by an upper root of descending fibres from beneath the corpora quadrigemina, and by a lower root of ascending fibres from a tract of grey matter which extends into the medulla oblongata (see p. 50), continuous with that from which the posterior cervical roots arise. The cutaneous distribution of the fifth nerve is continuous, on the head and neck, with these cervical roots, and the nuclear grey matter similarly continuous. This enables us to understand the radiation of pain from one nerve region to the other. The nucleus of the motor root is at the level of the origin of the nerve from the pons. The Gasserian ganglion lies in a hollow on the petrous part of the temporal bone, and thence the three divisions pass from the cranial cavity, the first by the sphenoidal fissure to the orbit, the second by the foramen ovale, and the third by the foramen rotundum, of the sphenoid bone to the spheno-maxillary fossa. The *first part* supplies the skin of the forehead and anterior part of the hairy scalp, the upper eyelid, and the bridge and tip of the nose; the *second part*, the lower eyelid, cheek, anterior part of the temples, side of the nose, upper lip, upper teeth, and upper part of pharynx, tonsils, soft palate, and uvula and roof of mouth; the *third part* supplies the rest of the temple, the anterior and upper part of the ear, the auditory meatus, the lower part of the cheek adjacent to the mouth, the lower lip, chin, lower teeth and gums, and the tongue, part of the mucous membrane of the mouth, and the salivary glands. The function of taste in the anterior part of the tongue is subserved by the lingual branch of the third division, but the fibres pass from this to the facial nerve, and thence to the spheno-palatine ganglion and the second division, as will be explained immediately. The motor part supplies the muscles of the lower jaw, temporal, masseter and pterygoid, the mylohyoid, and the posterior belly of the digastric.

The connections of the fifth nerve are numerous, and some of them are of considerable importance. The first part receives, at the Gasserian ganglion, fibres from the sympathetic, which pass with it to the eye, and are the fibres that innervate the radiating muscle of the iris. The second part gives off, from the spheno-palatine ganglion, the Vidian nerve, which (after a connection with the tympanic branch of the glosso-pharyngeal) joins the facial. It is called the large superficial petrosal, after it has given off a branch to the sympathetic. The lingual branch of the third part gives off the chorda tympani, which joins the facial in the Fallopian canal, a little below its junction with the large petrosal (Vidian). There is strong reason to believe that most of the fibres of the chorda tympani pass into the petrosal (Vidian), and thus reach the spheno-palatine ganglion and the second part of the fifth nerve. These fibres not only conduct taste impressions from the front of the tongue, but perhaps also subserve some tactile sensibility, since this has been lowered by disease of the chorda tympani. Lastly, the otic ganglion of the third part gives off the small superficial petrosal nerve, which is connected with the facial nerve where the Vidian joins it, and ends in the tympanic branch of the glosso-pharyngeal.

PARALYSIS OF THE FIFTH NERVE.

CAUSES.—The course of the fifth nerve renders it liable to damage from disease in various situations, but its deep position protects it from some influences to which nerve-trunks of superficial course are exposed, such as rheumatic neuritis, which is as rare in the fifth as it is common in the facial nerve. The chief causes of damage are the following:

(1) Disease within the pons, especially focal lesions, hæmorrhage, softening, tumours, and sometimes an islet of sclerosis at the level of the origin of the nerve, damaging either the root-fibres or the nuclei.

The most considerable symptoms are produced by damage to the root-fibres. The nuclear origin of the sensory fibres is so extensive that disease never affects more than a portion of it. Degenerative disease is rare; the motor nucleus usually escapes even in wide-spread nuclear degeneration.

(2) The nerve is often damaged by disease at the base of the brain, by tumours, chronic meningitis, especially of syphilitic nature, and caries of the bone. It is liable to suffer in disease of either the posterior or middle fossa, or of the petrous bone between the two. Inflammation may affect the root, primary, or secondary to a slowly growing tumour, and the resulting symptoms, in the latter case, may subside until renewed by pressure.

(3) Each division of the nerve has a course that exposes it to special lesions: the first, in the wall of the cavernous sinus, may be damaged by growths in the pituitary region, or aneurism of the internal carotid, and within the orbit it may suffer from growths or inflammation, such as orbital cellulitis; the second and third pass into the spheno-maxillary fossa, which is often invaded by tumours from the parotid region and adjacent bones.

(4) Traumatic injury, especially punctured and bullet wounds, through the mouth and nose. On the other hand, the nerve is rarely damaged in fracture of the skull.

(5) While secondary neuritis is common, arising by extension from bone or membranes, primary neuritis is rare, although now and then it is excited by cold, with or without a like affection of other cranial nerves, as the facial (*q. v.*). It may be predisposed to by gout, or caused by syphilis. Any part of the nerve may be thus damaged, and even the root may be inflamed through cold. A gouty man drove forty miles in a cold wind, and towards the end of the drive had transient double vision. Two days later the left fifth nerve became paralysed. In a case recorded by Ziehl* the neuritis apparently extended from the foramen ovale to below the junction with the chorda tympani. The peculiar neuritis that seems to be the cause of herpes zoster is frequent in this nerve, but must be distinguished from the ordinary form of inflammation. Interstitial neuritis has been found in hemiatrophy of the face (Mendel, &c.).

SYMPTOMS.—(A) *Sensory Portion.*—The chief symptom of an affection of the fifth or of its branches is loss of sensation in the region of the skin supplied by it, universal in severe disease of the trunk of the nerve, or when all three branches are damaged by a growth in the middle fossa of the base, but in disease of the branches limited to areas supplied by them. The loss of sensation is commonly preceded by symptoms of irritation, sharp, darting, burning pains referred to the region of its distribution, closely resembling those of neuralgia, and often accompanied by tender points in the course of the nerves. There may be increased sensitiveness, especially to pain. The duration of the stage of simple irritation varies according to the quick or slow progress of the disease; sometimes it is absent, and the anæsthesia is the first symptom. Tactile sensibility is usually lost first, and there is often sensitiveness to pain, when a touch cannot be felt. Ultimately both are lost. The muscles of the face are insensitive, but are not weakened, although the movements of the face have been observed to be a little slower than normal, apparently from defective sensation. The mucous membranes, as well as the skin, become insensitive. The conjunctiva can be touched and even pricked without discomfort or reflex action. The mucous membrane of the nose can no longer be irritated by snuff or ammonia. Odours can at first be perceived perfectly; after a time the sense of smell is blunted, in consequence of the dryness of the mucous membrane and secondary changes in its epithelial tissues. The anæsthesia extends over the mucous membrane of the lips, mouth, and tongue, up to the middle line. When the patient drinks, the cup, felt only on the unaffected side, may seem broken. Food is not chewed on the paralysed side because it cannot be felt, or because the muscles of mastication are weakened. Hence, fur accumulates on the anæsthetic half of the tongue: this accumulation has been ascribed to defective innervation, but it is common in all conditions which lead to one-sided chewing, and is probably merely due to the fact that the food no longer removes the epithelium. In some cases, at least, the back of the tongue, the anterior arches and the palate, soft as well as hard, are insensitive. In other cases, in which the extent of the cutaneous anæsthesia suggests disease of the whole of the fifth nerve, the loss of sensibility is confined to the anterior two thirds of the tongue. It is not known whether this difference is due to individual variations in the distribution of the nerve, or to differences in the seat of the disease, but it is certain that disease of the fifth *may* cause anæsthesia in the root of the tongue and palate.

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* 'Virchow's Archiv,' 117, 1889.

Another frequent symptom of disease of the fifth nerve is loss of taste. Disease of the root of the fifth nerve may cause complete loss of taste in all the gustatory region of the one side, tongue and palate. A syphilitic man, with paralysis of the left fifth and third nerves, had also complete loss of taste on that side; ultimately there remained only slight defect of sensibility on the cheek, weakness of the muscles of mastication, and loss of taste on that side, complete on the back of the tongue, partial in the front. Exceptions are probably cases either of partial disease of the root or of disease within the pons, where the taste-path has a separate course. Disease of the lingual, after the chorda tympani has joined it, causes loss of taste in the anterior two thirds of the tongue; above the junction with the chorda tympani disease of the third division seems to cause no loss of taste. Other facts are stated in the section on affections of taste.

Trophic changes may result from disease of the fifth nerve. Alterations in the vascularity of the face have been described, but are certainly rare. The secretions from the mucous membranes and special glands, lachrymal and salivary, are lessened in paralysis, and increased for a time in irritation. Rarely there is swelling and ulceration of the gums. An accidental bite of the insensitive cheek, for instance, heals slowly and tends to ulcerate. In long-standing cases the teeth have become loose. But the most important disturbance in nutrition is that of the eye. Inflammation of the eyeball is a common result of section of the nerve in animals, and has been frequently observed in man. The cornea becomes cloudy, then opaque, and ulcers form upon it, which may perforate and lead to a destructive inflammation of the globe. The conditions that determine this "neuroparalytic ophthalmia" (as it has been termed) have been the subject of much discussion. It is not simply the result of interruption of the sensory fibres, since such interruption, with complete anæsthesia, has been repeatedly observed without any ocular disturbance. In a patient under my care, complete paralysis of the fifth nerve, motor and sensory, has existed for seven years without a trace of ophthalmia. This fact, and the absence of inflammation in facial palsy, show that it cannot be due simply to irritation of the conjunctiva by foreign bodies, dust, &c., which are not felt, although such irritation may act as an excitant. From various experimental and clinical facts it seems probable that the inflammation is due, not to mere interruption, but to irritation of the fifth nerve, and is analogous to the acute trophic changes in the skin that depend on irritation of the sensory nerves (see vol. i, p. 22). An unsuccessful attempt to divide the nerve, which caused considerable irritation but no anæsthesia, has caused characteristic inflammation of the eyeball (Meissner), and electrical irritation of the ganglion causes ocular inflammation, which, although transient, is intense. It is probable, moreover, that such irritation is most powerful when it involves the Gasserian ganglion (especially the inner part, according to Meissner) or the nerve-fibres in front of the ganglion. It is frequently absent in disease of the nerve within the pons, and of its root at the surface of the pons, and although it may occur when the disease is in this situation, it is probable that a greater degree of irritation is required to produce it there than when the disease is at, or in front of, the Gasserian ganglion. Excision of the ganglion has caused such intense and uncontrollable inflammation as to necessitate the removal of the eye in a few days.*

Herpes zoster is frequent in the region supplied by the fifth nerve, especially in that of the first division. The observations of v. Bärensprung and others make it probable that the eruption is due to inflammation of the Gasserian ganglion, or of the nerve-trunks in front of this, although it is doubtful whether the cause is an ordinary neuritis. It is usually preceded or followed by much pain and hyperæsthesia, sometimes accompanied also by lessened tactile sensibility,—evidence of irritation and damage to the conducting nerve-fibres. In the old, the pain that follows herpes is often peculiarly enduring, and may last for months or years. Ophthalmitis or iritis may also occur. Herpes has been observed to follow other lesions of the fifth, but is not common in such cases, although it may attend the neuritis that is produced by cold. Catarrhal herpes of the lips has been attributed to neuritis of peripheral branches of the nerve, but without the pathological evidence that is desirable in the case of an eruption that differs so markedly from zoster in its irregular bilateral distribution and in its common cause.

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* Rose, 'Lancet,' Nov. 1st, 1890.

Facial hemiatrophy is certainly, as Mendel has shown, due in many cases to disease of the fifth nerve, but as we do not yet know that the relation is invariable it is described in a later part of this volume.

(B) *Motor Portion.*—The resulting loss of power in the muscles can be recognised by placing the finger on each masseter or temporal muscle, and making the patient bring the teeth forcibly together as in the act of biting. The feebleness or absence of contraction on the affected side is then evident. When the loss of power is slight, the affected muscles may contract a little later than the others. The paralysis of the external pterygoid causes two characteristic symptoms: the patient cannot move the jaw towards the unaffected side, and when the lower jaw is depressed it deviates towards the paralysed side, because, in depression, the external pterygoids draw the condyles forwards, and this movement occurs only on the unparalysed side. The mylohyoid and posterior part of the digastric act only with other muscles that are not supplied by the fifth, and hence their paralysis does not perceptibly impair the movement of the hyoid bone, which they help to raise. Although the tensor palati and tensor tympani are supplied from the fifth, no evidence of the paralysis of these muscles has been observed in cases of disease of this nerve. It has been assumed that the palsy of the tensor tympani would cause defect in hearing low notes, but I am not aware that the defect has been detected, and I have failed to discover it in cases that I have examined with special reference to this point. It is probable that the fibres to the tensor palati, although they may come from the fifth, are ultimately derived from the spinal accessory. After a time the paralysed muscles of mastication waste, the temporal and zygomatic fossæ become flattened, and ultimately a little secondary shortening of the muscles may limit, in a slight degree, the downward movement of the jaw.

Diagnosis.—The diagnosis of paralysis of the fifth nerve is easy when its degree is considerable, when the motor part is affected, and when the sensory part suffers without anæsthesia elsewhere, or with only paralysis of other cranial nerves. A diagnostic difficulty arises only when there is other sensory paralysis, or when the disease of the fifth causes only sensory irritation, and the resulting pain is like that of neuralgia. In hemianæsthesia the parts supplied by the fifth nerve, skin and mucous membranes, are insensitive, but so also are the back of the head, trunk, and limbs, and in many cases the special senses are impaired. As long as the only symptom of disease of the nerve is pain, due to irritation of the fibres, and referred to their distribution, the condition may be indistinguishable from ordinary neuralgia. The pain may be of the same character and seat, most intense in the same localities, and sometimes accompanied by the same tender points. Persistent hyperæsthesia of the skin is more marked and extensive in cases of organic disease than in neuralgia, and the pain radiates less frequently to other nerve regions, *e. g.* to that of the cervical plexus. In some cases there are peculiar abnormal sensations other than pain, and these are probably characteristic of slight disease of the nerve. The most conclusive evidence, however, is the development of anæsthesia, corresponding in range to the fifth nerve or one of its branches, which is proof of definite arrest of conduction. Another important symptom of such disease is loss of taste, which, as we have seen, may occur independently of anæsthesia. It should be carefully sought for in every case in which an organic lesion is possible.

The diagnosis of the seat of the disease depends on the extent of the symptoms, especially of the anæsthesia, and on the associations of the paralysis. When all parts are affected, the disease is commonly at the base of the brain or at the Gasserian ganglion. A lesion of the first part only is usually at the sphenoidal fissure, or in the orbit. If the symptoms are limited to the distribution of the second part, the disease is probably in the spheno-maxillary fissure, or the superior maxillary bone. Such symptoms are occasionally the first indication of a tumour of this bone. The third part is rarely diseased alone. An affection of the second and third divisions without the first is generally due to disease of the sphenoid bone or in the spheno-maxillary fossa.

The nerves to the eyeball are those most frequently associated with the fifth in disease at the side of the pons and in the middle fossa of the skull. In the former case all parts of the fifth suffer, in the latter only the first division. At the side of the pons the sixth nerve is more often associated with the fifth than any other, and next in frequency the third; disease of these nerves, and of all parts of the fifth, points conclusively to this position. Less commonly the facial and auditory suffer with the fifth. Paralysis of one fifth, and of the arm and leg on the opposite side, if of sudden onset, is due to a lesion within the pons; if of gradual onset, it may be due to disease in or outside the pons. An instance of the former (in which, however, arterial disease caused separate foci of softening) is shown in Fig. 53. Inability to move both eyes towards the side of the lesion is conclusive evidence of disease within the pons; and probably loss of taste, with palsy of the motor part of the fifth only, has the same significance.

TREATMENT.—The most important element, as in other cranial nerve palsies, is the removal of the morbid process as far as this is practicable; the special measures adopted must depend on its nature, syphilitic or other, as in the case of other nerves. When there is reason to suspect inflammation, a blister should be applied to the side of the occiput, or behind the ear, but not to the temple, lest it set up ulceration. If the affection has followed exposure to cold, hot fomentations may be applied to the side of the head and face, during the first three days, but not after the fourth day, or vesication may ensue. The pain is often very troublesome. Sometimes gelsemium relieves it. Cocain injected locally, by arresting peripheral impressions, may lessen the pain of organic irritation. Often, however, only hypodermic injection of morphia gives relief. A weak voltaic current occasionally lessens pain that is moderate in severity, but it is powerless over the more intense suffering. When there is anæsthesia without pain, stimulation of the terminal sensory fibres may be tried. If the continuity of the nerve-fibres is interrupted such stimulation is necessarily powerless, but in many cases of partial and stationary or regressive disease the fibres are slow in regaining functional activity, and this may be distinctly increased by their peripheral stimulation, which tends to overcome the resistance at the diseased spot. The best means of effecting this stimulation is the faradisation of the skin. A dry electrode influences the cutaneous nerves more than a moist sponge, and the wire brush is the most effective. The brush should be stroked over the anæsthetic areas, and the other electrode, which may be a wet sponge, should be placed behind the ear or at the occiput. The current should be strong enough, if possible, to cause some sensation, and it should be applied for two or three minutes. Faradisation is much better, for this purpose, than the voltaic current, since the latter, if strong enough to be effective, is apt to cause giddiness. Electricity is far better than irritating liniments, which readily set up trophic disturbance. The skin may with advantage be gently rubbed, so as to increase its vascularity, before the application.

As soon as there are any indications of disturbed nutrition of the eyeball, it should be kept covered to exclude the foreign bodies that aid in exciting inflammation. The occasional application of a drop of castor oil to the conjunctiva aids the subsidence of commencing affection. The success of such measures depends, however, on the degree of irritation of the nerve. When intense, the rapid inflammation baffles every attempt to arrest it, and exertion is needed to obviate the danger of a "sympathetic" affection of the other eye.

SPASM OF THE MUSCLES OF MASTICATION.

Spasm of the muscles supplied by the fifth nerve, the "masticatory spasm" of Romberg, may be either tonic or clonic.

Tonic spasm keeps the jaws together so that the two rows of teeth cannot be separated ("lockjaw"), or can only be separated for a short distance, a quarter or half an inch. Sometimes the teeth are pressed together with considerable force. The masseters and temporals can be seen to be prominent, and felt to be hard. An attempt to depress the lower jaw, and overcome the rigidity by force, causes pain in the muscles, and sometimes the spasm is itself painful. The muscular contraction is almost always bilateral. It is a conspicuous and early symptom in tetanus, traumatic and idiopathic, and is an occasional and late symptom in severe cases of tetany. It occurs also in hysteria; sometimes frequent brief paroxysms last a quarter of an hour or more, sometimes a more enduring form succeeds a hysteroid fit, and usually lasts until another convulsion, which leaves the patient free.* This spasm was unilateral in one hysterical case recorded by Travers.

In rare cases tonic spasm has been produced by sensory irritation elsewhere, generally due to injury, and has passed away when the irritation was removed. Romberg has recorded several instances of this. Unhappily, trismus, so produced, is in most cases the first symptom of general tetanus. This is true also of trismus following exposure to cold, but in one case paroxysmal and transient spasm of the jaw and tongue, accompanied by a "dying away of the extremities," was produced by any considerable exposure to cold (Romberg). In a few cases, prolonged trismus, without other symptoms, has followed similar exposure, but in most instances the patients were young women, and it is possible that the cases were hysterical in nature.

Another occasional and rare cause is irritation in the sensory region of the fifth nerve, due to carious teeth, ulceration in the mouth, and other causes. The tonic spasm has been observed to coincide with paroxysms of pain, and to be removed for a time by pressure on tender points in the branches of the nerve. It is said to be produced especially by irritation of the last molar, either from caries with alveolar abscess, or during the eruption of the tooth. In the latter case the spasm has been known to continue for several months (Germain).

Lastly, tonic spasm is an occasional symptom of organic disease of the pons, due to disease irritating, but not destroying, the motor nucleus of the fifth. The spasm may be unilateral, but it is more frequently bilateral (although sometimes greater in degree on one side) even when the disease is one-sided. It is often permanent.

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* There can be little doubt that the case recorded by Romberg ('Dis. Nerv. Syst.,' Syd. Soc. trans., vol. i, p. 305) of trismus after "epilepsy" was an example of this form.

A tumour is the most frequent cause of the symptom. Of recorded cases, in one (Marot) there was a small tubercle at the junction of the pons and medulla on the right side; in another (Wernicke) the tumour, also a tubercle, occupied almost the whole vertical extent of the left half of the pons, and had caused loss of the movement of both eyes to the left, paralysis of the facial nerve, extreme tension of the left masseter, and numbness of the right side of the head. Somewhat similar symptoms in a woman, aged forty-two, under my care, were probably due to syphilitic disease of the basilar artery; they developed in the course of two days, and then remained stationary. On account of the spasm of the muscles, which was bilaterial, the jaws could only be separated for a quarter of an inch; in this movement the jaw deviated quite the same distance to the left, and hence there was probably some weakness of the left muscles, although the voluntary contraction seemed equal. The sensory parts of the fifth nerve were unaffected, but there was entire loss of all lateral movements of the eyes, and limitation of the vertical movements; nystagmus, lateral in the right and rotatory in the left eye, which was strongly inverted; complete paralysis of all parts of the left facial nerve with degenerative reaction, and right hemiplegia, considerable in degree. Three years after the onset the spasm remained the same, and the other symptoms presented only trifling improvement.

Partial tonic spasm, affecting only some of the muscles supplied by the fifth nerve, is extremely rare. In the case of a girl suffering from hysteria and chorea, recorded by Leube,* the jaw was fixed for several days in the position of lateral deviation, and as there was no spasm in the masseters or temporals the symptom was apparently due to spasm in the pterygoid muscles of one side.

Clonic spasm of the muscles supplied by the fifth nerve is met with in two forms: (1) quick contractions frequently repeated; (2) single sudden contractions occurring at considerable intervals.

(1) The serial clonic spasm is the most common. It causes, if considerable, successive upward movements of the lower jaw, bringing the teeth together, sometimes with sufficient force to cause the sound popularly known as "chattering of the teeth."† If slight, there may be only a tremulous movement of the jaw, although the spasm of the muscles may be felt when the fingers are placed on them. The movement is almost always vertical; a lateral movement, from spasm in the pterygoids, has been described, but is extremely rare. Clonic spasm is bilateral in most cases. It is a conspicuous feature of many forms of general clonic spasm, as convulsion and rigor. It may occur also in paralysis agitans; in rare cases the muscles on one side may be affected before those on the other.

As an isolated symptom such clonic spasm is rare, and most cases have occurred late in life and in women. Romberg relates an instance in a woman aged sixty-five, who had previously suffered from facial spasm. The spasm affected the right masseter more than the left, and not the temporals. It caused a constant chattering of the teeth. Towards evening the movement became very forcible and violent; it ceased during sleep and also during mastication. Pain was felt only when the spasm was very violent.* I have met with one case in a woman aged fifty-five, in which similar spasm was associated with neuralgic "shooting" pain in the left side of the face, especially round the orbit, and about the malar bone, darting through the head, and sometimes along the lower jaw and down the neck. The pain was most intense in wet weather. The spasm appeared secondary to the pain, and was greatest when the pain was severe. Both masseters and temporals were involved. The spasm was not quite regular, but the frequency of the contractions was 72 to 80 per minute. At times it could not be felt. It ceased when the jaws were opened widely. It was rather stronger on the left side than on the right, and in gradually passing away (after some months of tonic and sedative treatment) it ceased on the right side sooner than on the left. Attacks of tonic and clonic spasm on one side, ultimately spreading to the tongue, have coincided with hemiatrophy of the same parts in a girl.†

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* 'Arch. f. klin. Med.,' 1869, vi, 273.

† Probably modified, by false analogy, from "clattering of the teeth."

(2) The second form of clonic spasm, in which there are occasional severe single contractions, sometimes occurs in chorea, but as an isolated affection it is very rare. The jaws are brought together with considerable force, and the tongue or cheeks are sometimes bitten. An instance of this form occurred in a woman aged twenty-six. It commenced suddenly; at first, whenever she tried to eat or began to talk, and also on going to sleep at night, the jaw would be suddenly jerked up and her tongue was several times badly bitten. She had no other symptom except slight weakness of the lower part of the face on the left side. She presented no evidence of hysteria. A heavy weight had fallen on the head about three months before the onset. The symptoms continued, decreasing in severity, for about a month, but for six months she had an occasional attack leaving tenderness in the masseters, and there was some reason to think that on one occasion in the night she had an epileptic fit. I have known similar spasms to occur during sleep, chiefly in men during middle life. They wake the patient, and the tongue, if it happens to be between the jaws, may be bitten. The affection appears to depend on enfeeblement of the nervous system, and ceases when this is strengthened.

DIAGNOSIS.—The existence of spasm in the muscles of mastication is readily recognised. The only conditions which simulate spasm are—(1) interference with the movement of the jaw by a tumour or inflammatory swelling near the ramus, conditions sufficiently obtrusive; (2) disease of the joint, usually rheumatoid, limiting the movement of the articular surfaces; here there is no tension of the muscles, and other joints are commonly affected. In one case, for instance, that of a man thirty years of age, in whom the lower jaw could not be depressed for more than half an inch, a similar condition had fixed the articulations of the cervical spine, so that the neck was as rigid as if it had been composed of wood.

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* Romberg, 'Diseases of the Nervous System,' Syd. Soc. trans., 1853, vol. i, p. 301.

† Sachs, 'Med. Record,' 1890.

The PROGNOSIS of isolated spasm is good, except in the cases which are due to organic disease of the nerve-centres. These are usually permanent. In other cases the spasm usually passes away, but often only after a somewhat prolonged treatment.

TREATMENT.—The treatment of the forms of spasm that are part of diseases of wider nature is necessarily that of the primary affection, and is described elsewhere. Hysterical spasm is sometimes removed by a blister behind the ramus of each jaw. In the isolated form it is important to search for and remove all sources of irritation of the sensory nerves, to lessen pain by sedatives, and to strengthen the nervous system by tonics, especially iron and quinine. When a carious molar is the cause of the spasm, it may be necessary to relax the muscles by chloroform or ether, that the tooth may be extracted. If the spasm is apparently due to cold, a hot air or vapour bath should be used in the early stage. Voltaic electricity has been said to do good in some cases—the positive pole being placed on the back of the neck, the negative on the contracting muscles, and if no result follows, the position of the poles may be reversed; but it is doubtful whether the current can reach the overacting centres. The paroxysmal form of clonic spasm is certainly lessened by bromides, and they usually prevent the occurrence of nocturnal spasm, but they have no influence on the tonic form. Cauterisation beside the cervical spine has been recommended for trismus due to cold (Petrone). In prolonged cases of tonic spasm in which the jaws are completely closed, the feeding of the patient is a matter of difficulty, and the extraction of a tooth may be needed for the purpose.

AFFECTIONS OF TASTE.

The sense of taste, it must be remembered, includes only the recognition of the qualities known as "bitter," "sweet," "sour," and "salt," with certain metallic sensations. It does not include what are called flavours; these, as we have seen, are really perceived through the olfactory nerve. Gustatory sensation is subserved by the mucous membrane of the tongue, palate, and palatine arches,* but in the forepart of the tongue the function is chiefly localised in the tip and edges, and is very slight on the upper surface. Each quality can be perceived in all parts of the gustatory region, but bitterness and sweetness are more readily appreciated at the back of the tongue, sourness and saltness at the tip and edges. Moreover, if minute areas are tested, there are found to be spots in which one quality can be perceived and not another. There seem also to be individual differences in the relative power of detecting the various qualities in the several regions. The sense is also influenced by age, being greater in the young than in the old.

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* Also on the posterior surface of the epiglottis, and even within the larynx (Michelson, 'Virchow's Archiv,' 1891, Bd. cxxiii).

In testing taste, substances should be used that do not appeal to any other sense,—colourless solutions or white powders. Care must be taken that their action is limited to the spot examined. If powders are used, a little time must be allowed for their solution by the moisture of the mucous membrane, and slight friction facilitates the stimulation. Salt, sugar, citric acid, and quinine answer very well; in spite of the slight solubility of solid quinine, its intense bitterness renders it readily perceived. The voltaic current affords a very convenient means of examination; it should be applied by two insulated wires twisted together, with exposed ends a few millimetres apart. A current of one or two cells causes a metallic taste wherever the function is intact. More cells should not be used, because pain is then produced, and obscures the sensation. The advantage of this test is that it influences directly the nerve-endings.

There is much difference of opinion as to the cranial nerve or nerves by which the sense of taste is subserved, and in connection with which its disorder should therefore be described. Hardly any question in physiology has been the subject of more discussion and of more experimental research. But, from its nature, the subject is one on which experiment can give no very certain sound, and has far less significance than observations on the effect of disease on man. Two separate questions are involved in the problem: first, the nerve-roots by which the taste-fibres pass to the brain; and secondly, the course of the fibres from the mucous membrane to those roots.

With regard to the first of these questions we have two important facts. First, taste has been abolished by disease of the root of the fifth nerve. There are many observations of loss of taste in the front of the tongue from this cause. One unequivocal case has been recorded by Erb, in which the fifth nerve in the middle fossa was involved in a mass of inflammatory connective tissue, and the nerves of the medulla were normal.[*] In most of these cases the state of taste on the back of the tongue has not been noted. In almost every case I have seen in which there were symptoms of disease of the root of the fifth nerve, outside the pons, I have found taste abolished on the corresponding side, not only on the front of the tongue, but also on the back of the tongue and on the arches of the palate.[*] On the other hand, cases occur in which there are symptoms of disease of the fifth nerve without loss of taste. It is probable that, in these cases, either disease of the root is partial and the taste-fibres escape, or else that the disease is within the pons, and that the path of the fibres for taste quickly separates from that of cutaneous sensibility, and so may escape when the latter is damaged. The latter hypothesis receives strong support from a case under my care in which there were symptoms of a small tumour within the pons near the level of origin of the fifth. In addition to palsy of the conjugate lateral movement of the eyes to the right, there was paralysis of the muscles of mastication on that side, and entire loss of taste on the same side of the tongue and palate, without any impairment of cutaneous sensibility. This case proves that the path of taste lies near and may be affected with the motor fibres or nucleus of the fifth, and without the fibres for cutaneous sensibility, and it indirectly shows the possibility of an affection of the latter without the path of taste. The second fact is that there is no recorded case in which the roots of the glosso-pharyngeal nerve have been diseased, and indeed no case in which there were symptoms of disease limited to the nerve-roots of the medulla, and in which there was any loss of taste.[†] These two facts constitute strong evidence that, in man, the fibres for taste reach the brain by the roots of the fifth nerve.

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[*] Erb, 'Neur. Cent.,' 1882, pp. 73 and 149. In a previous careful review of the subject ('Handb. der Krankh. der periph. Nerven,' 2nd ed., 1876, p. 220) he had come to the same conclusion, that the balance of evidence is in favour of the passage of the taste-fibres of the chorda tympani to the brain by the fifth nerve. Schiff has arrived at the same opinion from experiments on animals, and has summarised his conclusions in a lecture published in the 'Revue méd. de la Suisse Romande,' 1877, No. 1. Vulpian found the fibres of the chorda degenerated after

The peripheral path of the taste-fibres is, in part at least, strangely circuitous. It does not admit of doubt that the fibres from the front of the tongue are contained in the chorda tympani, which passes from the facial nerve to the lingual branch of the fifth. Apart from the results of experiment, it is only thus that we can explain the loss of taste in the front of the tongue that occurs in rheumatic facial neuritis, and in disease of the middle ear.[‡] Moreover, that these

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section of the fifth nerve within the skull, and not after section of the facial ('Gaz. méd. de Paris,' 1878, No. 19).

[*] One case, in which there had been loss of taste for several years, with palsy of the fifth, and afterwards slight weakness of the sixth, but no affection of the facial, auditory, or any other nerve, was published in the 'Journal of Physiology,' vol. iii, 1881. Since then I have met with many other cases. In all, the loss was proved by electrical examination as well as by the ordinary tests.

[†] In a case of fracture of the skull, and symptoms of injury to the nerves of the medulla, with loss of taste, recorded by Lehmann (Pflüger's 'Archiv,' xxxii, p. 194), direct injury to the nerves passing through the petrous bone was the probable cause of the symptoms. Cf. Ferguson, 'Med. News,' 1890.

[‡] Apart from the overwhelming evidence that has been accumulated, a crucial observation has been recorded by Urbantschitsch. In a case of disease of the membrana tympani and middle ear, causing loss of taste and some diminution of tactile sensibility in the front of the tongue on that side, chemical and mechanical

fibres run from the facial nerve towards the lingual, and are distributed to the tongue with the latter, is shown by the fact that section of the lingual for neuralgia, below its junction with the chorda tympani, has abolished taste.[*] But disease of the facial nerve within the skull does not impair taste; the fibres that reach the facial by the chorda tympani leave the nerve again, passing from the geniculate ganglion of the facial by the Vidian nerve to the spheno-palatine ganglion. Destruction of the Vidian nerve by an exostosis has abolished taste in the front of the tongue.[†] After reaching the fifth nerve they seem to ascend in the second division to its root and the brain.[‡]

Regarding the course of the taste-fibres from the posterior part of the tongue and palate, we have far fewer facts. Indeed, as regards man, the facts are practically limited to two: the loss from disease of the roots of the fifth nerve already mentioned, and the facts that taste is often lost on the back as well as on the front of the tongue in caries of the middle ear,[§] and has been totally abolished by a probable fracture of the base which paralysed the facial nerve on the same side.[‖] On the other hand, the distribution of the glosso-pharyngeal nerve to the "taste-buds" compels us to adhere to the view that the fibres subserving the function are distributed in this nerve. But it has connections with the fifth which may carry the taste-path to this nerve, and this affords the best explanation of the facts. The spheno-palatine ganglion (which supplies the mucous membrane of the palate and its arches) is connected with the glosso-pharyngeal by a branch from the large superficial petrosal to the tympanic nerve (of Jacobson), perhaps by a second, and from the otic ganglion the small petrosal goes to the same nerve. If the tympanic nerve of Jacobson and the chorda tympani are the two chief paths of the taste-fibres, their easy damage in disease or fracture explains the facts mentioned, while the proximity of the facial enables us to understand its simultaneous paralysis.[¶]

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stimulation of the chorda tympani produced sensations of taste and of touch on the part of the tongue in which the sensibility was impaired ('Archiv f. Ohrenheilk.,' xix, p. 135).

[*] Inzani, 'Meissner's Jahresbericht,' 1864, p. 555; Lussana, 'Arch. de Phys.,' 1871, p. 152; Mader, 'Cent. f. med. Wissensch.,' 1879, p. 395.

[†] Ferguson, 'Med. News,' Oct. 1890; the patient died from phthisis. He adduces additional evidence in support of the view that the path from the back of the tongue is by the tympanic plexus and the root of the fifth nerve.

[‡] In a case in which there were the symptoms of a lesion of the third division high up, above its junction with the chorda tympani, there was no loss of taste (Erb. 'Neur. Cent.,' 1882, p. 104).

[§] Urbantschitsch, 'Beob. über Anomalien des Geschmacks in Erkrank. der Paukenhöhle,' Stuttgart, 1876. I have several times satisfied myself of the occurrence of the loss of taste in these cases.

[‖] Bruns, 'A. f. Psych.,' xx. 1889. On the other side the fifth nerve was paralysed without loss of taste. This anomalous lesion suggests that the branches may have been damaged in different positions, and where they do not contain taste-fibres.

[¶] The nerve of Jacobson is that most frequently damaged in suppuration of the

We still need more facts as to the backward extent of the loss of taste from disease of the chorda tympani. In all observations on taste it is desirable that the three regions, front of tongue, back of tongue, and palate, should be specially tested. Opportunities for observation of the highest importance sometimes occur to surgeons, and it is very desirable that they should be utilised.

The peripheral nerve lesions that may cause loss of taste have been just mentioned. It may also occur as part of general hemianæsthesia, in disease of the cerebral hemisphere, and also, in the same association, in the functional disturbance of hysteria. When the loss is partial, the perception of some qualities may be more impaired than that of others, but there is never such complete loss of some perceptions without impairment of others as could be compared to colour-blindness. It is said to be sometimes due to local disease of the mucous membrane, but, on account of the wide area in which this function is subserved, the loss is probably never complete. The evidence of the loss is inability to perceive the qualities mentioned on p. 224. Its *treatment* is chiefly that of the disease causing it. If stimulation of the nerves is thought desirable, this can be readily effected by the voltaic current. One rheophore may be placed behind the mastoid process, and the other, a flat piece of metal, on the surface of the tongue. A tongue-depressor, insulated where it comes in contact with the lips, answers very well.

Perversion of the sense of taste, which has been termed *parageusia*, sometimes occurs in neurotic maladies, especially in hysteria and insanity, and also as an effect of the influenza poison, apart from catarrh. It has also been observed in tabes, associated with perversion of the sense of smell, but distinct from it. In this condition a substance gives rise to some other sensation than that which it ordinarily causes. The symptom is often associated with morbid dislike to certain tastes. *Increased sensitiveness*, *hypergeusia*, is said to be met with occasionally under the same conditions as perversion of the sense, and so also are *subjective sensations* of taste, usually of an unpleasant character. These are also met with as a result of irritating disease of the nerves, such as disease of the ear, and they have been experimentally produced by electrical stimulation of an exposed chorda tympani. Such sensations sometimes occur as the aura of an epileptic fit, and as part of the hallucinations of the insane. They must, of course, be distinguished from actual sensations due to abnormal buccal secretions, and from those due to the presence in the blood of substances that can stimulate the gustatory nerves. The various subjective sensations or perversions of taste scarcely ever call for special treatment.

middle ear, and next the chorda tympani, according to Godsderm, of Copenhagen (1888).

FACIAL NERVE.

The facial nerve, *portio dura* of the seventh pair, has a tortuous course both within the pons and through the wall of the skull. Its deep origin has been described at p. 49. At the surface of the brain and within the internal auditory meatus the facial and auditory nerves lie together and suffer together in disease. From the auditory meatus the nerve has a winding course through the temporal bone, passing first outwards to the inner wall of the tympanum, and then backwards above the foramen ovale, and downwards behind the tympanum to the stylo-mastoid foramen. It lies throughout this course in the bony "Fallopian canal," but the layer of bone which separates it from the cavity of the tympanum varies in thickness, and may even be incomplete. Moreover two nerves pass from it into the cavity of the tympanum, the small nerve to the stapedius muscle,* and the chorda tympani, which leaves the facial a few millimetres above the lower opening of the canal, and courses across the upper part of the tympanic cavity and membrane, to pass again through the bone and join the third division of the fifth. An arterial twig also passes from the canal into the tympanum. These connections are important, since the nerve is often damaged by disease of the middle ear, and they afford considerable and variable anatomical facility for this damage. At the bend in the temporal bone there is a gangliform enlargement on the nerve, the "geniculate ganglion," from which the large petrosal nerve passes (as the Vidian) to the spheno-palatine ganglion; and, as already stated (p. 214), there is reason to believe that most of the fibres from the chorda tympani leave the facial by this nerve and reach the brain through the fifth. The gangliform enlargement also receives a twig from the nerve (small petrosal) which connects the otic ganglion, through the nerve of Jacobson, with the glosso-pharyngeal. Outside the skull the nerve gives branches to the occipital part of the occipito-frontalis, to the external ear, to the stylo-hyoid and digastric, and then divides, opposite the posterior edge of the masseter, into numerous branches which pass to all the muscles of the face, and to the platysma myoides beneath the skin of the neck.

The path from the facial nucleus to the cerebral hemisphere crosses the middle line of the pons above the nucleus, so that if the face is paralysed by one-sided disease above the middle of the pons, the paralysis is on the side opposite to the lesion, as in ordinary hemiplegia. The further course of this path has been described at p. 75. It is probable that the bilateral muscles of the upper part of the face are also connected with the hemisphere of the same side.[*]

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* It is not unlikely that the fibres for the stapedius come from the small "intermediate nerve of Wrisberg," since this arises from the accessory auditory nucleus.

FACIAL PARALYSIS.

Paralysis of the face results from any interruption to the path from the cortex to the muscles. The character of the paralysis differs according as the disease involves, on the one hand, the path above the nucleus (which we may term *supra-nuclear palsy*), or, on the other hand, the nucleus itself or the fibres of the nerve, whether within the pons or outside it (*nuclear and infra-nuclear palsy*). In the latter case all parts of the face are affected, the orbicularis palpebrarum and frontal muscle, as well as the muscles of the mouth; but in the former the upper muscles of the face are little or not at all affected, and the muscles which go to the angle of the mouth suffer chiefly. Another difference is that in supra-nuclear paralysis voluntary movements are often more impaired than emotional movements; in nuclear and infra-nuclear disease they suffer equally.

Lastly, there is an important difference between the two forms in the reaction of the nerve and muscles to electricity. In supra-nuclear disease the reaction is normal, or presents only a trifling change, which is the same to the voltaic and to the induced current. Acute lesions of the nucleus or nerve, unless trifling in degree, cause secondary degeneration of the fibres, with loss of all irritability, and preservation only of the voltaic irritability of the muscle-fibres.

It is customary to term paralysis from disease of the nerve "peripheral;" that from disease of the nucleus or root-fibres in the pons is also sometimes termed peripheral, because it resembles that from disease of the nerve; sometimes it is termed "central;" paralysis from disease of the path above the nucleus is also sometimes termed "central," sometimes "cerebral." The latter term should be used only with the understanding that a similar paralysis may occur from disease of the crus or upper part of the pons. Moreover the word "peripheral" is sometimes applied to disease of the extremities of the fibres. Paralysis from disease of the convolutions is termed "cortical." Otherwise the nomenclature given above is preferable, disease of the root-fibres being termed "radicular."

The supra-nuclear palsy has been already considered in the chapter on hemiplegia; we have now to consider only that which results from disease of the nucleus, the root-fibres, or the nerve.[†]

CAUSES.—(1) Within the pons the nucleus or root-fibres may be damaged by various focal lesions, usually in association with the motor tract of the opposite limbs, or with the sixth nerve of the same side, round the nucleus of which the facial fibres course (Fig. 38, p. 50). In very rare cases of anterior polio-myelitis (infantile paralysis), the facial nucleus suffers; I have seen one instance of this. Primary degeneration rarely affects all parts of the facial nuclei, but the nerve-cells related to the orbicularis oris often degenerate with those for the hypoglossal nerve, in chronic "labio-glossal paralysis." Diphtheritic palsy involves the face in rare cases, and probably consists in a subacute affection of the cells of the nucleus and the fibres of the nerve.

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[*] In the rabbit, medial section of the pons paralyses the elevator of the upper lip on both sides, even to stimulation of the cortex (Exner and Paneth, 1888).

[†] It is often termed "Bell's paralysis," after Sir Charles Bell, who first explained its nature.

(2) At the base of the brain the nerve may be compressed by tumours or damaged by meningitis or hæmorrhage. The auditory nerve is usually affected with it.

(3) Within the canal in the temporal bone the nerve suffers in various forms of otitis and bone disease, especially in children. In most cases there is caries of the bone and suppuration in the middle ear,[*] and the disease of the bone between the Fallopian canal and the tympanum extends to the nerve. This may be merely inflamed, or may be destroyed by the spreading disease. The amount of ear disease that exists before the facial nerve is affected varies much, and the readiness with which the nerve suffers doubtless depends in part on the thinness of the protecting lamina of bone. It is said that the nerve may be paralysed when only the lining membrane of the tympanum is inflamed and the bone is not diseased. I have known paralysis to occur when caries was certainly limited to the mastoid bone. In such cases we must assume an extension of inflammation along the chorda tympani, the nerve to the stapedius, or the arterial twig already mentioned, unless the layer of bone is imperfect. It is perhaps by such extension that facial paralysis has sometimes followed sore throat—simple, or such as occurs in some acute diseases.[†]

(4) Injury. Fracture of the base of the skull often passes through the petrous bone, and the nerve may be torn or bruised. The face is then at once paralysed, but in some cases the nerve is damaged only some days (three to five) after the injury, and the mechanism must then be a secondary process, probably inflammation.[‡] Meningeal hæmorrhage is possibly a rare cause, especially that which occurs from the cerebellum during birth; this may explain some "congenital" cases in which it has been associated with weakness of the neck due to damage to the spinal accessory nerve.[§] Outside the skull, the nerve or some of its branches may be divided by wounds about the ramus of the jaw, and especially in operations for the removal of tumours in the parotid region. A simple blow on this part may also damage the nerve. An angry schoolmaster struck a boy with the corner of a book just below the ear. Much pain followed about the ear and the side of the head; in a day or two facial paralysis came on, com-

[*] It is not, however, a frequent consequence of caries, although the calculation of only 1 per cent. made by Bezold is perhaps too low (Bezold, 'Labyrinth-Necrose,' 1886).

[†] Wendt, 'Archiv der Heilkunde,' 1870.

[‡] See Demoulin, 'Gaz. méd. de Paris,' 1888.

[§] Stephan, 'Rev. de Méd.,' 1887.

plete and, as it turned out, permanent, in spite of all treatment. The blow cost the schoolmaster a thousand pounds. Facial paralysis is sometimes, although rarely, caused during instrumental delivery, by the pressure of the blades of the forceps on the nerve over the jaw, and such paralysis has even been produced on both sides. Compression by the shoulder has also been effective.*

(5) Neuritis is the cause of the common facial paralysis, due to cold, and often termed "rheumatic." The majority of cases of facial paralysis seem to be of this nature; and although, often, no special exposure to cold can be traced, there has then been usually some habitual exposure. The features of these cases are so uniform, allowance being made for differences in degree, that we are justified in regarding the pathological condition as the same in all—a neuritis within the Fallopian canal.† The inflammation perhaps affects chiefly the sheath of the nerve, which swells and compresses the fibres, expansion outwards being prevented; but in a case in which the actual state could be observed the nerve sheath was not distinctly inflamed;‡ the external restraint may indeed cause the inflammation to spread into the nerve, and in this case the fibres were extensively degenerated. For a long time a very different explanation was given. It was supposed that cold acts by paralysing the terminations of the facial nerve in the muscles. But there is no evidence that cold ever paralyses intra-muscular nerve-endings, and we know that paralysis of all fibres of one nerve, and of no other fibres, always means disease of the trunk of the nerve.

Inflammation was found also in a perfectly similar paralysis, due to a different cause, recorded by May.§ The patient was suffering from leucocythæmia (an affection which may cause lymphoid neuritis), and the nerve within the Fallopian canal presented a fusiform swelling three tenths of an inch long, due to an infiltration of lymphoid cells; the nerve-fibres at the spot were in process of destruction.

In more than half the cases a special and considerable exposure to cold can be traced; generally a draught of cold air has blown on the side of the face and head, as in sitting in a railway carriage opposite an open window, standing at an open door, driving in a cold wind. One patient had ridden a bicycle during three cold nights; another had lingered on a seaside pier in a cold wind; in another the exposure to cold occurred the day after a beard, worn for thirty years, was shaved off. The symptoms usually commence within two days of such exposure. In other cases there has been more or less constant exposure, as by working in a draughty shop. Occasionally the sufferer presents other evidence of rheumatism, especially of the fibrous tissues, and a liability to a similar affection of other nerves, as in the cases mentioned on p. 144. But many of the subjects of facial palsy present no other evidence of rheumatism, and the affection may be an isolated event in an otherwise healthy life. In a few cases there is a history of gout, inherited or acquired, which may have predisposed to the disease; but it is, on the whole, very rare to obtain evidence of this. In some instances there is other evidence of catarrh, general fever, sore throat, or neuralgic pain in the neighbourhood of the affected nerve. The neuritis sometimes arises by distinct extension from external cellulitis; in one case the cellulitis was produced by chronic eczema of the skin in front of the ear, over the place of emergence of the nerve.* In other cases such cellulitic swelling is evidently catarrhal; in one it existed beneath each jaw for a week before the onset of one-sided palsy.

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* Varnier, 'An. de Gyn.,' 1888.

† It has been conjectured that the inflammation sometimes affects the nerve after its emergence, but no evidence of this (*e.g.* swe ling of the nerve) has been noted.

‡ Minkowski, 'Berlin. klin. Wochenschr.,' 1891.

§ E. May, 'Aerztl. Intell. Blatt.,' 1884, No. 31.

Grouping together all cases which may be due to Fallopian neuritis (excluding those that are the result of ear disease or syphilis), they appear to be rather more common in males than in females, no doubt from the greater exposure of the former. Of 112 unselected cases of which I have notes, 66 were in males, 46 in females. Rarely more than one member of a family suffers—as, for instance, a man and his niece. Such coincidences are doubtless sometimes accidental. The affection may occur at any age, but is rare under ten. I have met with it at seven, and twice in the second year of life. It is most common between twenty and thirty (27 cases), and is equally frequent in each of the other decades between twenty and fifty; after fifty it becomes rare, but is occasionally met with even in old age (cases at seventy-four and seventy-seven). Of 80 cases due to this cause, 46 occurred between October and March inclusive, and 34 between April and September.† It thus occurs at all seasons, but is rather more frequent in winter than in summer.

(6) Syphilis is an occasional cause, but its frequency may readily be over-estimated if the mere fact of preceding syphilis or of improvement under iodide of potassium is admitted alone to be evidence of nature. We are only justified in regarding the paralysis as syphilitic when there is or has been other evidence of active syphilis, and when there is no other recognisable cause; the syphilitic nature of the case is more probable if the nerve is affected within the cranium. In

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* It has been thought by some that cold affects the facial nerve by causing a catarrhal inflammation of the middle ear, which spreads to the nerve. But this seems improbable when we consider how frequent catarrh of the middle ear is without facial paralysis, and how rare are obtrusive signs of tympanic inflammation when the nerve is affected, and that slight inflammation of the tympanum may be produced coincidently with neuritis by cold without the former being the cause of the latter. Earache often follows the onset, but very seldom precedes it even for a day, and early pain is more common in other situations.

† Twelve cases occurred in January; 10 in November; 9 in June; 7 in each month, May, August, October, December; 5 in February, March, and in April; 3 in July and in September. In the third of the year, October to January, 36 cases; February to May, 22; June to September, 22.

many cases of facial paralysis in the subjects of syphilis, the paralysis is distinctly excited by cold, but it is possible that the syphilis may have had a predisposing influence. The mechanisms by which syphilis causes facial paralysis are by meningitis, by a gumma on the nerve-sheath or near it, and probably also by neuritis.

(7) Primary hæmorrhage into the nerve-sheath or Fallopian canal is a rare cause.* There are cases in which facial paralysis comes on in a few minutes and is at once complete, and in which there is no indication of any central disease. Such cases, of which I have seen five, may be due to an extravasation into the Fallopian canal or the nerve itself, disposed to, perhaps, by some peculiarity of vascular arrangement. Thrombosis in a vessel of the nerve is also a conceivable mechanism, especially when sudden palsy occurs under conditions favouring this lesion—as, for instance, in one case, a week after childbirth. It affords a possible explanation of the curious cases in which such an onset follows emotion. Complete paralysis occurred quite suddenly in a lady who was at the time watching the dressing of a huge wound in her own breast, produced by the destruction of a cancer by caustic. In one instance the onset was an hour after a sudden fright; in another it coincided with a sense of faintness.

(8) Facial paralysis has been observed in rare cases of tabes, in which its cause resembles doubtless that of the other nerve palsies that are met with in the disease. The nerve may also be paralysed by an islet of disseminated sclerosis within the pons, when others affect the spinal cord.

Hysteria may coincide with facial palsy, especially since the latter is not uncommon in young adults. The coincidence has no real significance; although the cases have been needlessly described as "hysterical," they present no feature to warrant the epithet, except in so far as hysterical contraction lessens movement.

Double facial paralysis is rare. Its causes are (1) disease of the pons Varolii; possibly a lesion in the middle of the pons, where the two facial paths cross; certainly lesions on both sides of the pons, such as the bilateral softening that is produced by disease of the basilar artery. (2) Disease, syphilitic or other, of the nerves at the base of the brain; by this usually other nerves are also damaged. In one case of this kind, in which there was also simultaneous paralysis of both auditory nerves (not aural), the onset occurred after exposure during sleep to a cold wind, but the patient had had syphilis. In another case only one auditory nerve was involved. (3) Simultaneous double otitis.† (4) In paralysis from cold, both sides of the face, as they are occasionally affected at different times, may, in extremely rare cases, suffer at the same time.‡ In multiple neuritis the face is sometimes involved, always on both sides, and generally in association with the limbs as part of a wide-spread affection. It is met with chiefly in certain rare forms of polyneuritis, due to special toxic blood-states, and not in the common variety, produced by alcohol. In diphtheritic paralysis, in which polyneuritis is the chief element, double facial palsy sometimes occurs. (5) Partial paralysis (of the lips on both sides) occurs from nuclear degeneration in labio-glossal paralysis, or symmetrical cortical lesions, and (6) a peculiar form of facial palsy associated with palsy of the eye muscles, and due probably to nuclear wasting has been already mentioned (p. 197). It may be congenital or come on during infancy.

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* Wilks and Moxon, 'Path. Anat.,' p. 257, and Moxon, 'Path. Trans.,' vol. xx.

† Wright, 'Brit. Med. Journ.,' Feb. 27th, 1869. A week separated the onset of the palsy on the two sides. The patient was a man aged sixty-five.

‡ Kruge, 'Neur. Cent.,' 1890.

Symptoms.—In complete facial palsy the muscles of the affected half of the face become toneless and immobile. In all movements, voluntary or emotional, the affected half of the face is still. The two sides of the face present a strange incongruity, and the smile or frown, deprived of half its range, loses more than half its character, so that it is difficult to recognise the expressional significance of the distorting contractions of cheek and brow which occur on the unaffected side. The influence of the loss of tone on the position of the features at rest differs according to the age of the patient; the smooth features of youth are largely moulded by the elasticity of the skin, and are little changed by the paralysis of the muscles; slight drooping of the angle of the mouth may be the only sign of paralysis during rest. But it is otherwise when time has scored the face with furrows. These are due to the loss of elasticity of the skin, and their position is chiefly determined by the muscular tension that is caused by, and expresses, the emotions dominant in the past. With the loss of this tension the furrows change their place or vanish, as the flaccid skin adjusts itself in obedience to other influences (Fig. 103). The effect of the age-changes is seen conspicuously in the forehead and lower eyelid. The transverse furrows of the forehead cease suddenly at the middle line. In the young the lower eyelid is held by the elasticity of the skin almost as close to the eyeball as on the unaffected side. In the old it falls forwards, and the tears, increased by the irritation of the unprotected eye, cannot reach their proper duct, and the eye "waters." But in young and old the loss of movement is the same. The affected side takes no part in

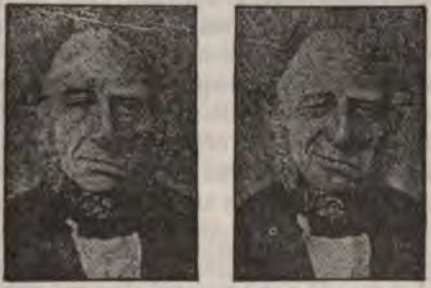

Fig. 103.—Facial paralysis in a man aged sixty-five, showing the influence of the loss of tone on the inelastic skin. The figure on the right represents an attempt to close both eyes. (From photographs.)

frown or smile. The eyelids cannot be brought together; in a strong effort to close them, the eyeball is rolled upwards, so that the cornea is beneath the upper lid, and only the sclerotic is visible. The patient often imagines, ostrich-like, that his eye is shut, because the cornea is covered. During sleep the eye remains partly open, because normally closed not only by relaxation of the levator, but by contraction of the palpebral part of the orbicularis. But the upper lid follows, in a normal manner, the downward movement of the eyeball, since the orbicularis takes no part in this descent, except in keeping, by its tone, the lid in contact with the eyeball. In the old, the lower lid, on looking up, does not rise so well as on the other side, apparently because the lid is not kept in sufficient apposition to the globe. In smiling and other movements the mouth is drawn towards the unaffected side, the zygomatic muscles being unopposed by their fellows. From the displacement of the mouth the tongue is protruded to one side of the orifice, and thus may seem to deviate even when it is exactly in the middle line of the face. The lips cannot be pressed together on the paralysed side, and hence the air cannot be so compressed within the mouth as to be expelled in a puff. The patient accordingly finds that he cannot blow out a candle. In drinking, the liquid runs out of the paralysed corner of the mouth, and the patient has to incline his head to prevent this. Whistling is, of course, impossible, and smokers may first discover the paralysis by finding that the ejected saliva takes an inconveniently erratic course. Speech is frequently a little changed by the imperfect articulation of the labial consonants. The palsy of the buccinator permits food to get between the teeth and the jaws, and it is found more convenient to chew upon the other side. The palsy of the stylo-hyoid and posterior belly of the digastric does not cause recognisable symptoms. The platysma myoides is paralysed, as may be observed by making the patient depress his lower lip, an action in which the platysma contracts. The dilator naris being inactive, the nostril does not expand on sniffing, and may even yield to the pressure of the air; smell, in consequence, is less acute than on the other side. The muscles of the external ear are also paralysed in those persons in whom these muscles were under the influence of the will. All reflex movements are of necessity lost when the paralysis is due to disease of the nerve-fibres or their nucleus—an important distinction from cerebral palsy, in which they are preserved.

Palate.—It has been said that the palate is sometimes paralysed on the same side as the face from disease of the facial nerve, and it has been supposed therefore that the levator palati is supplied by fibres which pass from the geniculate ganglion of the facial, by the large petrosal nerve and Vidian, to the spheno-palatine ganglion. But the opinion that the palate ever suffers from disease of the facial nerve seems to be erroneous. The levator is supplied from the spinal accessory (*q. v.*), and in more than a hundred cases of facial paralysis, due to disease of the nerve in various situations, carefully examined, I have never observed a corresponding defect of movement in the palate. Deviation of the uvula was occasionally observed, but its inclination was as frequently from as towards the paralysed side. Such obliquity of the uvula is not uncommon in those who present no facial palsy, and sometimes is considerable in degree. When present in facial palsy its state is unchanged after the face has recovered, and it is not associated with any defect in the movement of the palate. In rare cases in which there has been actual coincident paralysis of the palate there was probably a simultaneous affection of the spinal accessory nerve or of its palatine branch. In most, other nerves have suffered, and this may be damaged not only at the medulla, but outside the skull, by deep-seated cellulitis just below the place of emergence of the facial. Of this I have seen some well-marked instances, with and without implication of the other parts of the nerve, or of the hypoglossal.*

Taste.—In disease of the facial nerve between the origin of the chorda tympani and the geniculate ganglion, taste is lost in the anterior part of the tongue on the affected side. It is not lost when the lesion is of the root of the facial nerve,† or of the nerve-fibres within the pons. It is lost in about half the cases of paralysis from cold, doubtless because the neuritis extends up the nerve to the origin of the chorda. When the nerve is damaged outside the skull, taste is generally unaffected; the exceptions are probably due to an ascending neuritis. The affection of taste may persist after the paralysis, but usually passes away before it. Sensation in the face is generally unaffected, but I have several times found in early and severe cases a very slight diminution in the sensitiveness of the skin on the motionless side, especially on the cheek. The cause may be an alteration in the function of the nucleus of the fifth nerve, due to the diminution of the impressions that are normally produced by muscular action. This is more probable than that the facial nerve sometimes conveys sensory fibres from the skin.‡ I have also occasionally found (in early cases) an area of anæsthesia on the front and back of the concha, in the region of skin supplied by a nerve given off by the facial as it emerges, and which is probably derived from the fifth nerve.

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* Dr. Hughlings Jackson has also stated that he has never seen paralysis of the palate due to disease of the facial nerve ('Lancet,' April 2nd, 1887). He made this remark in connection with an instructive case of paralysis of the face and palate, which at first sight seemed to prove the association, but further investigation showed conclusively that the palsy of the face was of cerebral origin.

† Certain recorded cases of disease of the root and loss of taste, opposed as they are to the usual rule, are explicable on the assumption that the descending degeneration was attended, as it sometimes is, by descending neuritis, and that this invaded the taste-fibres at or below the geniculate ganglion.

‡ Vaso-motor disturbance, œdema, and increased sweating have been observed, probably also of secondary relation or due to accidental association of an affection of the sympathetic (Frankl Hochwart, 'Neurol. Centralbl.,' 1891).

Hearing is impaired in cases of facial paralysis due to the extension of inflammation from the bone or tympanic cavity, and also when the facial and auditory nerves are affected together by basal disease. Impairment is occasionally met with in cases due to cold, but is slight unless there is also a catarrh of the middle ear. Tinnitus often attends the onset, and is probably due to the same cause; it may continue for weeks. When the disease involves the middle part of the nerve in the canal the stapedius is paralysed, and the tensor tympani, being unopposed, often increases the sensitiveness of the ear to musical notes, especially to those of low tone (Lucae). A subjective high-pitched sound has been referred to the same cause.

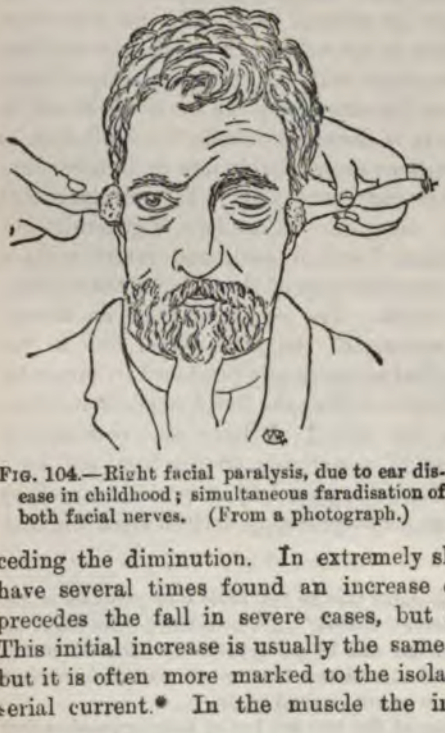

FIG. 104.—Right facial paralysis, due to ear disease in childhood; simultaneous faradisation of both facial nerves. (From a photograph.)

The electrical reactions in facial paralysis are those characteristic of peripheral palsies,—the degenerative reaction fully described at vol. i, p 54. The charts there given are from cases of disease of this nerve. In a severe case the nerve rapidly loses its irritability to faradic and voltaic stimulation and this may be extinct in the course of ten days or a fortnight. In slighter cases the irritability of the nerve is lowered but not lost; a strong current still excites it. In most cases the fall in irritability does not commence for three or four days after the onset of the paralysis, and in slight cases may be delayed for a week or ten days. It may be distinct in the nerve-trunk some days before it is found in the muscles, *i. e.* in the intra-muscular nerves. During the first few days an increase of irritability may often be found preceding the diminution. In extremely slight disease of the nerve I have several times found an increase corresponding to that which precedes the fall in severe cases, but followed by no diminution. This initial increase is usually the same to both forms of electricity, but it is often more marked to the isolated faradic shock than to the serial current.* In the muscle the initial increase may also be recognised, and depends on changes in the intra-muscular nerve-fibres. This increase is followed by a gradual fall in the faradic irritability of the muscles, corresponding in degree to that of the nerve, and due to the fact that the faradism only stimulates the muscles through the intra-muscular nerve-endings. With this loss of faradic irritability, that to the voltaic current is preserved and increased; to it the muscle-fibres themselves respond, and respond with undue readiness, so that they contract with a weaker current than is required by the muscles of the unaffected side. The form of response to increasing currents is often (but by no means always) changed, and instead of 1 KCC;* 2 ACC, AOC; 3 KOC, we may have such order as the following, which I take from my notes of actual cases: 1 KCC = ACC; 2 AOC; 3 KOC or 1 ACC; 2 KCC, AOC; 3 KOC or 1 KCC, AOC; 2 ACC; 3 KOC. A continuous contraction (tetanus) is also readily produced during the passage of the current, as in 1 KCC; 2 ACC = KC Tet.; 3 AC Tet.

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* It is said that in the slightest cases there may be no change in irritability, and the statement is doubtless true. But in the cases that I have seen in which the irritability to both currents was normal, I have seldom failed to find a distinct increase of irritability to the isolated induction shock. An increase, with sluggish effect, was persistent for months in a somewhat anomalous case observed by Koch (Hosp. Tid, 1889).

With the recovery of the nerve the irritability of nerve and muscle slowly returns to the normal, but the nerve-fibres often recover some conducting power (*i. e.* the paralysis lessens) before they regain their local excitability. For a long time this remains below the normal. The muscles lose their excessive degree of irritability before the order of response becomes normal.

In rare cases the reaction in nerve and muscles is that of the mixed form (the "middle form" of Erb), in which, with only slight diminution in the irritability of the nerve, the muscles present the increased and qualitative change characteristic of degeneration. The varieties in the course of the reactions are illustrated in the charts in vol. i (Figs. 35—43).

The electrical reaction presented by one muscle, the orbicularis oris, merits special mention. The muscle sometimes illustrates in a striking way the increased but slow muscular response to the voltaic current which results from nerve degeneration. The voltaic current diffuses itself widely; applied to the angle of the mouth on the unaffected side the closure of the circuit causes a quick sharp contraction in the normal half of the orbicularis, distinctly *followed* by a slower contraction on the paralysed side. If the strength of the current be then reduced until it is too small to cause contraction in the normal muscle, it may still cause the sluggish contraction on the opposite, paralysed, side. This has been mistaken for a reflex contraction; it is no doubt the result of the diffusion of the current.

In cases in which the paralysis of the face remains absolute, the paralysed half of the orbicularis usually regains some power. This is doubtless due to the fact that, the muscle being circular, the two halves are continuous, and the nerve influence from one side extends beyond the middle line. It is indeed probable that the nerves of the two sides join, and some regeneration from the sound side may be possible. I have occasionally found, in old-standing cases, otherwise complete, that faradism applied to either angle of the mouth caused contraction of the whole of the orbicularis—a fact difficult to explain on any other hypothesis than that of nerve regeneration from the healthy side.

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* KCC, kathodal closure contraction; KOC, kathodal opening contraction; Tet., tetanic contraction (see vol. i, p. 56).

Muscular wasting follows the nerve degeneration in the face as elsewhere, and in those who have but little subcutaneous fat the loss of tissue may be perceptible. But in most cases the atrophy is not revealed by any change in the contour of the face, because the thin muscles constitute but a small part of the subcutaneous tissues, and the nutrition of these is unaffected. True facial hemiatrophy never results from disease of the facial nerve.

The loss of the protective movements of the eyelids exposes the conjunctiva to various irritant influences, but inflammation is limited to slight conjunctivitis. The increased secretion of tears, although a source of annoyance to the patient, doubtless supplements the defective action of the lids in removing foreign particles. In the old there is occasionally a slight conjunctivitis of the lower lid, induced by the exposure which is the consequence of its recession from the eyeball (see p. 217). I have once known a small ulcer to form on the cornea, but it quickly healed.

The characters of the facial palsy are not influenced by either the seat or character of the disease of the nerve. In partial lesions some fibres may suffer more than others, with a corresponding variation in the relative degree of paralysis in the several parts of the face. But paralysis due to disease of the nucleus presents an important difference from that which is produced by disease of the nerve-trunk. The fibres for the orbicularis oris are dissociated from the other facial fibres in their nuclear origin, and are connected with the fibres for the tongue; the two suffer together in nuclear degeneration (labio-glossal paralysis). On the other hand, in the case of facial palsy from nuclear inflammation (part of polio-myelitis), the orbicularis oris had escaped entirely, although the rest of the face was absolutely paralysed.*

Certain concomitant symptoms occasionally attend the onset of facial paralysis, and are due, for the most part, to the cause of the nerve-lesion. In neuritic cases, pain about the ear is very common, and may be felt for a day or so before, but is more common after the onset. It may be like "earache," usually slight, but is more frequent behind or below the ear, and may extend down the neck or along the ramus of the lower jaw or below it. In the latter situations it is often accompanied by tenderness. It sometimes extends over the whole of the corresponding side of the head, and radiates to the face;

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* The connection between the fibres for the orbicularis oris and the tongue is very close (see note on p. 50).

there and in the neck it may have a neuralgic character, and sometimes lasts for weeks. More often its duration is brief—one or a few days. A little swelling in front of the ear may often be found, if looked for, in the early stage; it is sometimes considerable and extensive, and is no doubt due to cellulitis. Auditory symptoms have been already mentioned. In one case the patient complained, at the onset of the paralysis, of a phosphorus-like taste in the back of the tongue, explicable, on the hypothesis stated at p. 227, by irritation of the nerve of Jacobson or the tympanic plexus. Slight giddiness not unfrequently attends the onset, and may be due to a disturbance of the labyrinth; it is not due to paralysis of the stapedius. Sometimes the giddiness is intense, especially when there is disease within the skull, probably from injury to the nerve-fibres from the semicircular canals, or to the middle peduncle of the cerebellum. In some cases there are the signs of more wide-spread disturbance, nervous or general, from the exposure to cold that causes the paralysis. Beside severe neuralgia in the head or face or neck, I have met with transient dilatation of the pupil and derangement of the movements of the eyeball on the same side, and also on the other side; ptosis on the other side, hyperæsthesia of the side of the tongue and gums and also of the cheek, and an eruption of herpes on the surface of the tongue on the side of the affection of the face. These various nerve affections must be due to independent associated effects of cold.* Coincident catarrhal fever is met with rarely, sore throat occasionally, and even transient albuminuria.

Course.—The onset of facial paralysis is usually rapid but rarely sudden. The palsy commonly reaches its height in from four hours to two days. Sometimes the affection comes on during the night; it is slight in the evening and complete next morning; often it is trifling on first rising, and becomes considerable or complete in the course of the day.

Its duration varies according to the degree and character of the nerve-lesion, which may be severe or slight from any cause. In the slightest form the paralysis may last only for a week or ten days, but often the affection lasts for months, and two or three months is the average in cases of moderate severity. Those of greater degeee last for six or eight months, and it is very common for the ultimate recovery to be incomplete. Indeed, whenever there is nerve-degeneration, and whenever the paralysis is complete for a month, some trace of it usually remains, although this may be seen only on close scrutiny. In rare cases no recovery takes place.

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* The herpes on the tongue has been referred to the chorda tympani; but this is unlikely, because it may precede or coincide with the onset of the affection of the facial nerve, and may exist when taste is unimpaired. The peculiar hyperæsthesia on the side of the tongue and gums, mentioned above, preceded the facial palsy for a week (the cause being exposure to cold), and, from its distribution, must have been due to an affection of the lingual. Herpes zoster of the cheek, such as must be ascribed to the fifth nerve, has been observed as a coincident effect of cold (Strübing, 'Arch. f. kl. Med.,' 1885).

Secondary Over-action.—In all severe cases some muscular contracture comes on, usually as voluntary power returns. When the palsy remains absolute the contracture is absent; the muscles remain toneless. The first trace of contracture usually manifests itself about four or six months after the onset of the paralysis; it slowly increases for eight or twelve months, and then remains stationary or lessens. It is most marked, at rest, in the zygomatic muscles, and reproduces the lost naso-labial wrinkle, often in deeper degree than on the other side, so that on looking at the face the first impression is that the healthy side is the weaker. The impression may seem to be confirmed by a slight movement of the face, for this is more marked on the affected side than on the other.* But if the movement is considerable, the side that moved first is seen to move much less than the other. With this contracture is constantly combined a tendency to associated over-action. A smile is, in health, accompanied by a slight contraction in the orbicularis palpebrarum, and this contraction is increased on the affected side, so that the eye may almost close. Conversely, when the eyes are closed by a strong contraction of the orbicularis, the zygomatici on the affected side over-act and draw outwards the angle of the mouth (Fig. 105). The contractions may also be readily excited by reflex action through the fifth nerve. In many cases another symptom is added after a time—spontaneous twitchings, isolated spasmodic contractions, recurring at irregular intervals, and affecting chiefly the zygomatici. These various spasmodic symptoms are often considerable when the amount of regained power is slight.

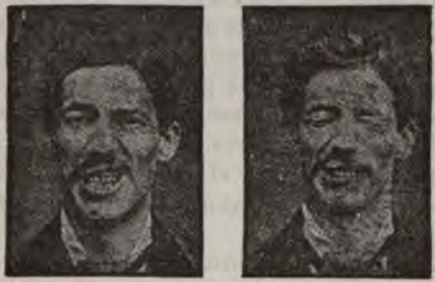

FIG. 105.—Old paralysis of the right side of the face, late contracture and associated over-action. The figure on the left shows an attempt to raise the upper lip; that on the right, closure of the eyes. In the latter it is seen that while the patient's right eye is closed less firmly than his left, the naso-labial furrow is rendered much deeper on that side by the associated over-action of the zygomatici. (From photographs.)

The effect of the muscular contracture on the aspect of the face at rest varies according to the age of the patient, in a manner opposite to the effect of the palsy. In the old it reproduces the lost naso-labial furrow, and restores the symmetry to the lower face. In the young it develops a furrow that has no counterpart on the other side, and so adds a deformity at rest to the distortion during movement, and is a source of annoyance scarcely less than the original palsy. It may cause the tears to flow on to the cheek, or force them into the nose with undue speed so that an increased nasal secretion is simulated, and perhaps a real excess of lachrymal secretion is sometimes induced. The condition is very persistent. When slight, and succeeding a trifling paralysis, it may pass away in time, but when considerable, and after a severe paralysis, it usually lasts unchanged for years, and although it may subsequently lessen, it rarely passes away entirely. I have known it to be still considerable twenty-five years after the attack of facial palsy. It has been attributed to the use of electricity in the treatment of the palsy; faradism and voltaism have both been blamed, but the contracture and over-action develop in as marked degree in patients who have not had electrical treatment as in those who have. Its probable cause will be presently considered.

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* It is possible that there is not, as there seems, an undue initial movement, but that the previous contracture renders the effect of an equal voluntary contraction more conspicuous on the affected side.

Second attacks of facial paralysis, due to cold, are rare, but I have met with eight instances. In three the second attack was on the same side, and in all the first attack was slight. The second attack was from three weeks to four years after the first. In most cases it was distinctly excited by a fresh exposure to cold. I have also twice known a slight facial paralysis, which was improving, to relapse. Cases have been met with in which there were more than two attacks, and these have been termed "recurring facial palsy," and compared with the recurring paralysis of the third nerves occasionally met with. Five instances are recorded by Bernhardt: in four, the same side was affected several times; in the other case the side varied several times in the series of attacks. The precise nature of such cases is not known; they probably constitute a special form.*

PATHOLOGY.—The evidence of the nature of these cases has been already stated, and the general pathology of such disease has been described in the first volume. But our knowledge of the special pathology of the affection is meagre. We do not know why this nerve suffers with such frequency, nor have we any adequate information regarding the elements of the nerve that are primarily inflamed. The curious absence of pain in the affected region of the nerve has been already mentioned (vol. i, p. 67).

The cause of the late over-action and spasm is probably a change in the functional state of the cells of the nucleus of the nerve, produced by their altered functional condition during the complete paralysis, but, once established, permanent. The contracture always coincides with some recovery of power; in a case of absolute and lasting paralysis there is no contracture. Since the associated over-action and the clonic spasm accompany the contracture, it is reasonable to refer all to the same cause. The tonic contracture and associated over-action are excessive degrees of normal phenomena, and the latter must be due to the connection between the groups of cells. The resistance in the cells and between them becomes lessened, so that they act with undue readiness on each other and on the fibres. Their tonic influence on the muscles is increased, the action of one part of the nucleus spreads too widely through it, and the cells are liable to spontaneous discharge.* The excited cells are imperfectly discharged through their fibres during the palsy, the axis-cylinders being, it will be remembered, really parts of the cells, and not mere effluent channels. Another mechanism of derangement may be the loss of the afferent impulses from the muscles during their palsy. These (from the facts stated at vol. i, p. 204), must lower the abnormal activity of the stimulated cells, because their deficiency will interfere with the due limitation of the excitation, and promote the diffusion among the cells of the nucleus, of the activity which should influence only certain fibres and certain muscles. Once established, the morbid action tends to perpetuate itself, and is hence most intractable.

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* Bernhardt, 'Berlin. klin. Wochenschr.,' 1889.

Diagnosis.—The existence of facial paralysis is only too conspicuous as soon as the state of the muscles is revealed by voluntary or emotional action. But when the face has partly recovered, and is the seat of late contracture, it is easy to mistake the side affected, unless attention is paid to the strength of contraction, and the significance of the associated over-action is remembered. A slight movement is more conspicuous on the affected side; a strong one is far less in degree. It is well also to remember that patients are apt to misstate the side affected in consequence of their frequent impression that the smooth unwrinkled half of the face must be natural, and that the side which is distorted by movement is unnatural. Often no reliance can be placed on the statements made by patients or their friends as to the side affected.

The distinction of paralysis due to disease of the nerve-fibres from that due to disease of the motor tract from the hemisphere (infranuclear and supra-nuclear palsy) rests especially on the distribution of the paralysis, as stated on p. 79. The most important guide is the condition of the eyelids. If there is such persistent paralysis of the eyelids that they cannot be perfectly closed, the lesion is probably in the nucleus or nerve-fibres. Cortical disease may for a few days paralyse the orbicularis, but other symptoms are usually present in a cerebral lesion, and the different course of the two affections soon renders the diagnosis clear. Moreover, emotional movement is less impaired by cerebral disease than is voluntary movement, or

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* The hypothesis, so far, is essentially the same as that put forward by Jacobi ('Inaug. Diss.,' Marburg, 1877) in regard to a case of similar spasm after palsy of the nerves of the arm. Hitzig has suggested that the disease of the nerve leads to a state of "irritation" in the centre, but the significance of this statement depends upon its meaning.

vice versâ; in disease of the nerve the two are equally affected. In the latter, reflex action is lost; in the former it is unchanged.

In nuclear and infra-nuclear disease the reaction of degeneration is found in the nerves and muscles; in lesions of the facial path above the nucleus the reaction is normal or nearly so. Thus the degenerative reaction is proof of a lesion in the nucleus or nerve, but a normal or nearly normal reaction does not prove that the disease is above the nucleus, since it occurs also in very slight lesions of the nerve.

The further diagnosis of the seat of the lesion in the nerve rests on the associations of the paralysis. If no nerve except the facial is affected, and taste is unimpaired, the lesion is probably in the lowest part of the Fallopian canal or outside the skull. If taste is lost on the front of the tongue, the lesion is in the canal, and involves the nerve between the geniculate ganglion and the origin of the chorda tympani. It does not, however, prove that the disease began in this part; disease may spread to the chordal region. Moreover, taste may be lost for a time, afterwards becoming normal although the face remains affected; its loss is often unknown to the patient, and needs to be sought for. Paralysis of the palate was formerly thought to show disease of the geniculate ganglion or nerve above it, but it is not produced by paralysis of the facial nerve. Slight deafness is of little localising value; complete deafness, without ear disease, coming on at the same time as the paralysis of the face, indicates disease in the internal auditory meatus or at the base of the brain, commonly the latter, since disease within the meatus is extremely rare. Paralysis of the facial and sixth, without the auditory, is generally due to disease in the posterior part of the pons, where the facial fibres curve round the nucleus of the sixth nerve. It is possible, however, as I have seen, for the sixth nerve to be paralysed by a simultaneous rheumatic neuritis. If the orbicularis oris is quite unaffected, and all other parts of the nerve are paralysed, the disease is within the pons; if the orbicularis only is paralysed and the rest of the face is free, the disease is in the neighbourhood of the hypoglossal nucleus, and the tongue is almost certain also to be involved.

PROGNOSIS.—The cause of the paralysis is of less prognostic significance than are its characters. The chief etiological indication is that the prognosis is good in *pure* syphilitic cases, in the paralysis from pressure during birth, and (if the patient lives) in paralysis from the causes of polyneuritis, including diphtheria. It is not good when the onset is so sudden as to suggest hæmorrhage into the nerve. It is said to be better in ear disease if the tympanum is not perforated (Dalby). But in ear disease and all other causes, especially in those due to cold, cases of slight and severe character are met with, and the prognosis must be mainly founded on the electrical reaction. It is true that signs of improvement, and its rate, may enable an estimate to be formed of the likelihood of recovery, and of the probable duration of the affection; but if, as is often the case, there is no recovery of power when we have to form an opinion, we should have no prognostic guide were it not for the information electricity affords us. By its means we can ascertain the state of function of the nerve-fibres, and infer the state of their nutrition, and thus we can gain more information than even the microscope could supply. If at the end of ten days the irritability of the nerve is not below the normal the face will probably be well in a few weeks. If, on the other hand, at the end of a fortnight the irritabibity of the nerve is absolutely lost, the paralysis will certainly last for several months. If at the end of a fortnight the nerve irritability, although lowered, is not lost, recovery will probably occur in about two months. Between these forms, intermediate gradations of severity occur, for which the prógnosis must be correspondingly modified. When the faradic irritability has been absent for some weeks, any rëturn of excitability in the nerve-fibres indicates a speedy return of some power in them.

Treatment.—The first element in treatment is to arrest and remove, as far as possible, the morbid process that is damaging the nerve. If there is even a possibility that this may be syphilitic, iodide of potassium should be given, and if there is no speedy improvement, mercury should be added. If there is ear disease, free exit for any discharge should be secured. When exposure to cold is the apparent cause, or in similar cases in which no cause can be traced, if the paralysis has existed for a few days only, fomentations should be applied to the region in front of and below the ear, as hot as can be borne, for half an hour, and repeated every three hours for two or three days. In all cases, except those of very trifling degree, a blister should be applied behind the ear, over the mastoid process if the nerve is diseased in the canal, or on the side of the occiput if it is at the base of the brain. In rheumatic cases, the patient should take a smart purge and avoid alcohol. An alkaline diuretic mixture (bicarbonate of potash, or acetate of ammonia, and nitrous ether) may be given at the onset, together with small doses of mercury during the stage of active inflammation. If there is evidence of general rheumatic catarrh, a hot bath or, better still, free diaphoresis may be employed. It is important that the patient should, if possible, keep indoors. If this is impossible, the side of the head should be protected. Subsequently the blister may be repeated, and small doses of iodide of potassium may be combined with the mercury, and tonics, as quinine and strychnine, may be given later.

There is no evidence to show that electricity has any influence over the process of nerve degeneration or recovery. But the voltaic current excites the muscles to contraction when they are otherwise absolutely inert. It is reasonable to conclude that such functional stimulation of the muscles helps to keep up their nutrition. The nerve-fibres may recover functional power after months of interruption. During this time the muscles, left alone, undergo partial atrophy, and it is probable that this is less if galvanism has been sedulously employed. If a case has not been galvanised, after several months of absolute paralysis scarcely any contraction may be obtained the first time the current is applied, but after a few applications the muscles may respond vigorously, and it sometimes happens that the first indication of returning voluntary power follows such a "waking up" of the muscles. The positive electrode should be placed below the zygoma and the negative stroked along the course of the muscles, along the zygomatici, the orbicularis, in the upper and lower lip, the levator anguli oris, across the frontalis and the eyelids, the upper eyelid being lowered. The strength used should be the minimum to which the muscles will respond; usually from four to eight cells of an active battery are sufficient, the current used for the eyelids being weaker than for the other muscles. It has been said that the positive electrode should be applied to the muscles, because these respond more readily to it than to the negative pole. But this is not always the case; sometimes the response to the negative pole is the earlier; often it is the same to each pole, and the irritability to the negative is seldom much behind that to the positive. The rule given above is, therefore, on the whole the best. The application should be made for a quarter of an hour once or twice a day. It can be made perfectly well by the patient himself, seated before a looking-glass. As the face recovers, the increased voltaic irritability lessens and faradic irritability returns, slight at first, but gradually increasing. It has been advised that faradism should then be substituted for voltaism, but it is very doubtful whether faradism does good. The voluntary power which is then regained constitutes a stimulus to the muscles far greater and better than that of faradism, and it influences the same muscular fibres. If any electrical treatment is continued it is better to still use the voltaic current, which not only stimulates the nerve-fibres as surely as faradism, but also any muscular fibres of which the nerves have not yet recovered. When there are indications of commencing late contracture, the use of faradism is distinctly contra-indicated, and it is perhaps better to stop all electrical treatment that causes discomfort. Although this is certainly not the cause of contracture, it may yet tend to increase it, and when this stage is reached electricity has probably effected all that it can achieve.

For the contracture and over-action little, unfortunately, can be done. I have never been able to observe any beneficial result from either drugs or local treatment. The use of a weak unbroken voltaic current has been recommended, but its influence is inappreciable. Warm douches, or steaming the face, and the internal administration of bromide of potassium have been recommended (Rosenthal). Daily gentle shampooing of the face, by moving the finger, with gentle pressure, along the course of the muscles from origin to insertion, may be adopted as having an influence in the right direction, and at least incapable of doing harm. Faradisation of the muscles of the sound half of the face neither has nor can have any influence on the contracture, beyond a tendency to increase it by reflex stimulation through the fifth nerve.

FACIAL SPASM.

Spasm in the muscles supplied by the facial nerve may, as we have just seen, be secondary to a paralysing lesion, but it occurs also apart from preceding paralysis, and this primary form alone is here considered. It is sometimes termed "*mimic spasm*," from the semblance of emotional expression which results, and also, after the French, *convulsive tic.**

Facial spasm may affect only a few muscles or almost all those of one side, and either form may be unilateral or bilateral. It may be due to organic disease, or "idiopathic," depending on functional and nutritional changes. The spasm is usually clonic; if there is tonic spasm there is almost always clonic spasm as well.

The orbicularis palpebrarum and zygomatici are more prone to spasm than other muscles of the face; the motor mechanism for the orbicularis muscle is the more sensitive, in consequence of its important function and energetic reflex action, while both muscles take the leading part in emotional expression. Hence spasm, ultimately general, often commences in one of these, and partial spasm affects them far more frequently than any other part of the face. Tonic spasm limited to the eyelids, is termed "blepharospasm," and will be separately described; while the habit-spasm of childhood, a totally different disease, is considered in connection with chorea.

Idiopathic spasm is met with only during adult life, after twenty years of age. That which succeeds paralysis alone occurs in childhood and youth. Ordinary facial spasm usually commences between forty-five and sixty, rarely earlier, but sometimes later. The earliest typical case I have met with commenced at thirty-two, but cases are on record in which it began between twenty and thirty. Two thirds of the cases I have seen commenced between fifty and sixty. An inherited neurotic tendency to insanity or epilepsy in relations can sometimes be traced; direct heredity is very rare, but I have known a mother and daughter to suffer, each in late life.† Women suffer far more frequently than men, and constitute about two thirds of the cases. It does not seem to have any special relation to hysteria; the tonic contraction of the eyelids or quivering of the face sometimes met with in this disease is quite different from ordinary facial spasm. A general neuropathic tendency is common, and many sufferers have been liable to periodical headaches. Anæmia and general defective nutrition of the nervous system conduce to its occurrence, but the cause of the idiopathic form that can be most frequently traced is mental anxiety, sometimes a sudden shock, more often prolonged grief or concern. In this connection it is instructive to note, on the one hand, the intensely disturbing influence of such emotion on the nervous system, and, on the other, the relation of contraction in the facial muscles, and especially of those in which the spasm often begins, to emotion: this cause lowers the nutrition of the nervous system, and may have a special tendency to derange the structures on which it chiefly acts. In many, the disease begins about, or soon after, the climacteric period. In one case it existed during the whole of two successive pregnancies, ceasing on delivery, but afterwards coming on without this cause.

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* This is the original meaning of the word "tic," concealed in part by its use as an abbreviation of the more common *tic-douloureux* (neuralgia) to which it was applied, either metaphorically, from the twitch-like character of the pain, or else on account of the spasm that may accompany severe paroxysms of neuralgia. The original French form *ticq* is supposed by Skeat to be allied to ***tukken*, Low German *to twitch*** (modern German *zucken*).

† Rosenthal has described the affection of five members of a family, but he does not state the form of spasm. In most cases of direct inheritance the affection is not true facial spasm, but a kind of habit-spasm.

Another occasional cause is irritation of the fifth nerve, to which the facial has a special reflex relation. The irritation may be in the eye, in the teeth, or through the cutaneous branches of the fifth nerve. But it may survive the irritation which sets it up; it followed a fortnight's severe toothache on the same side, in one instance, but did not cease when extraction of the decayed teeth relieved the pain. Another patient had been liable, all her life, to attacks of migraine, with severe pain in the eyeball around which the facial spasm began. Rarely neuralgia in the neck has preceded the spasm, but neuralgia, however violent, seldom sets up spasm of independent course, in spite of the fact that muscular contraction often accompanies paroxysms of severe pain. Irritation of the intestines or uterus has been alleged as a cause, but on insufficient evidence. Severe cold acting on the face and head has apparently caused the disease in a few instances, perhaps also by the reflex mechanism of irritation of the fifth nerve, since in some of these cases, although not in all, pain has preceded the spasm. It is to be noted that the effect of these influences is not always immediately manifest. Thus a man's face was scorched, and his eyelashes and eyebrows were burned, by the explosion of a cartridge; a month later, when the face was well, spasm commenced in the muscles of the eyelids and nose, and continued for years. A soldier during the Franco-German war suffered from severe neuralgia of the right side of the face, induced by cold; two years afterwards this side of the face became the seat of spasm (Bernhardt). In such cases we must assume that the irritation induces a susceptibility in the facial centres which permits slighter influences to cause a morbid activity. Such susceptibility must also be assumed in the cases, considerable in number, in which no cause can be traced.

Two other causes give rise to special varieties of the affection. First, it may develop out of some habitual movement, as in a man who was in the habit of taking snuff on the affected side. Allied to such cases, but still more closely to habit spasm, are the cases that may be called "senile habit spasm," in which there is some frequently repeated movement of the face. In a man of fifty-five, strong momentary contractions of both orbiculares recurred continually, and were ascribed to working by a strong light. In another, more closely allied to the typical cases, facial spasm develops from torticollis by extension from the neck muscles. In this also two causal varieties may be distinguished. When the primary spasm is in the muscles of the back of the neck on both sides, the frontales contract simultaneously, raising the eyebrows. This is the result of the normal association which is readily observed when a person looks upwards. When the spasm is in the muscles moving the head to one side, it may spread to all the muscles commonly affected in facial spasm on that side. If such spasm in the neck is bilateral, both sides of the face may be involved. Such extension to the face seems to occur when the spasm inclines the head towards the shoulder, rather than when it merely rotates it.

Organic disease causing facial spasm has been in one of two situations; in the facial nerve, or the facial centre in the cortex of the opposite hemisphere. In several cases spasm has been produced by a tumour pressing on the nerve at the base of the brain, a small

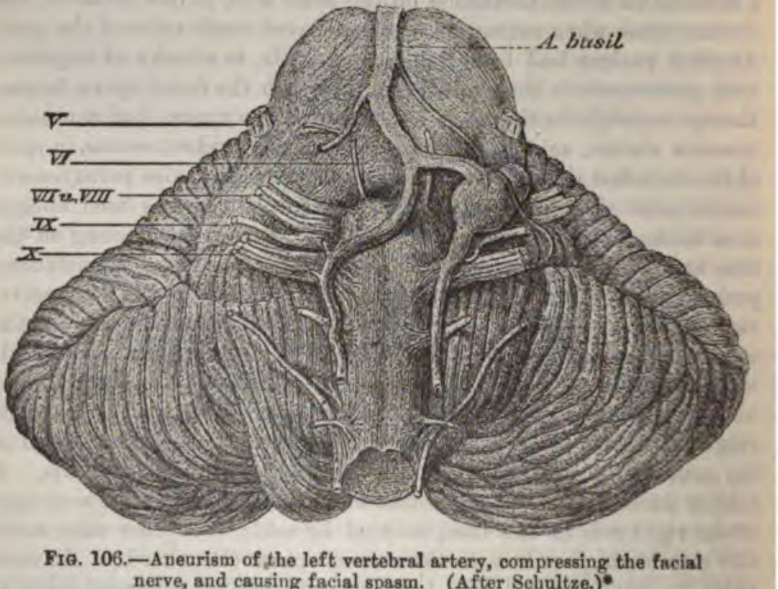

FIG. 106.—Aneurism of the left vertebral artery, compressing the facial nerve, and causing facial spasm. (After Schultze.)*

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* The patient was a man, aged fifty-six, who had received an injury to the head ten years before. For a year before death he had suffered from short quick contractions in the left side of the face, increased by any movement of the jaws or

sarcoma,* a cholesteatoma (Schuh), or tumour of the cerebellum (which, however, also compressed the pons, &c.), a tumour of the auditory nerve close to the pons,† or an aneurism of an adjacent artery. An instance of the last, an aneurism of the vertebral, is shown in Fig. 106. Spasm does not seem to be caused by disease in the petrous bone, in spite of the frequency with which the nerve suffers in this situation, and it does not seem to be caused by neuritis except as a sequel to paralysis. In organic lesions of the pons, spasm has only been observed as a transient symptom, preceding paralysis, in consequence of an abscess.

On the other hand, persistent facial spasm has been produced by a small stationary lesion of the ascending frontal convolution in the position of the facial centre, opposite the inferior frontal sulcus. Fig. 107 shows a point of softening in this situation, which caused persistent clonic spasm, limited to the zygomatic muscles.‡ It has also been the first symptom of a tumour in this situation.§ I have seen one case in which it was highly probable that facial spasm was the result of a cortical injury during birth. In other cases the spasm has

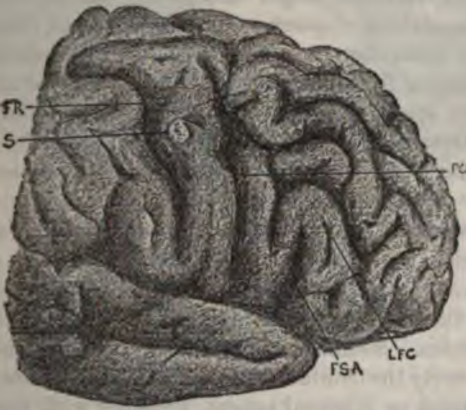

FIG. 107.—Small focus of superficial softening, S, in the ascending frontal, opposite the origin of the fissure between the middle and lower (L F C) frontal convolutions, which had caused persistent clonic facial spasm. P C S, pre-central sulcus; F R, fissure of Rolando; F S A, anterior limb of fissure of Sylvius. (From a photograph kindly furnished by Dr. Berkeley, of Baltimore.)

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face. All muscles were affected except the frontalis; the palate did not move. There was no pain. No morbid changes could be found in the nerves on naked-eye or microscopic examination (Schultze, 'Virchow's Archiv,' Bd. lxv, p. 385). Compare also a case narrated by Buss ('Neur. Centralbl.,' 1886) in which the posterior cerebellar artery was dilated.

[*] Moos, 'Archiv f. Augenheilk.,' 1874, Bd. iv, Abth. 1, p. 179.

[†] Sharkey, 'Brain,' 1888.

[‡] Berkeley, 'Medical News,' July 15th, 1883. The case recorded by Allen Starr, 'American Journal of Med. Science,' 1884, Case 51, is the same.

[§] Walton, 'Boston Journal,' 1889.

followed a fall on the head; and a slight injury to the surface of the brain on the opposite side, such as often results from contusion, is highly probable. This may readily have occurred even when the spasm is on the side of the skull which received the blow. But the cases in which there is reason to suspect an organic lesion, in any situation, form only a small minority.

SYMPTOMS.—In some cases of facial spasm the muscles of one side of the face present frequent momentary contractions, resembling those produced by the stimulation of the nerve with the faradic shock. In others there is a single contraction, which recurs at irregular intervals of seconds or minutes. More frequently there are brief paroxysms in which there is both tonic and clonic spasm, and the latter sometimes has a quick quivering character. In other cases again the paroxysms are longer and more violent, lasting several minutes, and consisting of quick contractions, two or three per second, with imperfect relaxation between them, and occasional prolonged contractions, lasting five or ten seconds. A paroxysm may begin and end with clonic spasm, while the chief part of it consists of tonic contraction. Occasionally the attack is interrupted by brief periods of complete relaxation, or complete except in the orbicularis palpebrarum.

The orbicularis and zygomatici are generally more affected than the other muscles, so that the eye is half closed, the angle of the mouth is drawn outwards, and the naso-labial furrow is deepened. In a few cases the elevator of the upper lip is more affected than the zygomatic muscles. Sometimes the spasm involves the corrugator supercilii, but rarely the frontalis, although this is occasionally involved in either one-sided or bilateral spasm, together with the orbicularis; the contraction in either may preponderate, so that the eyebrow is raised or lowered during the attack. Both orbiculares or corrugators or frontales may be involved, although the spasm is in other muscles one-sided. The depressor of the angle of the mouth, the levator menti, and the platysma myoides are sometimes implicated. The last may even become hypertrophied from the continued over-action (Fig. 108). The effect of the preponderant contraction in the orbicularis and zygomatic muscles is a curiously mixed emotional aspect, a sort of whimpering smile. The orbicularis oris is scarcely ever involved.

The spasm may be confined to the muscles of one side, and is almost always so confined at first. It usually begins gradually, and is at first slight and occasional, often occurring only under some exciting influence. In the orbicularis palpebrarum it may be at first so slight as to resemble the familiar quivering popularly termed "live blood." It may cease for a time, and then return in more persistent form. Often in the severer paroxysms there may be slight spasm on the other side, of the eyelids or angle of the mouth, or of the levator menti, and in rare cases, after a time, the spasm becomes altogether bilateral. Spasm limited to the muscles about the eyes is, indeed, often bilateral, involving chiefly the orbiculares, sometimes the corrugators, but it is exceptional for the lower muscles to be equally affected on both sides.

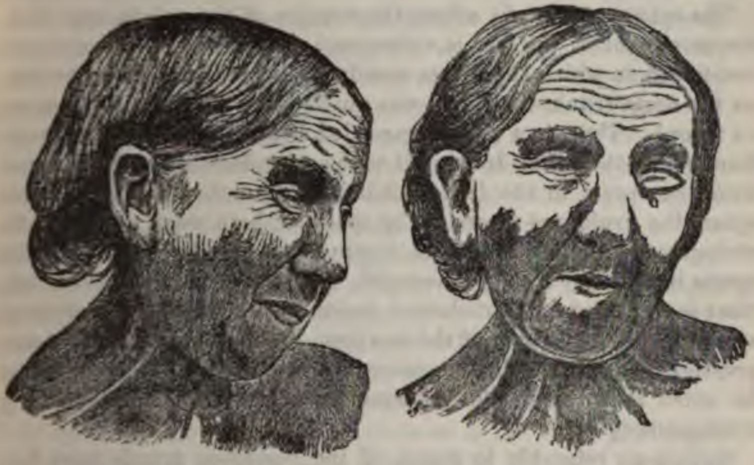

Fig. 108.—Bilateral facial spasm in a woman of sixty-one, due to grief. Over-action and apparent hypertrophy of platysma.

The spasm is usually lessened by rest, physical and mental. It is always increased by emotion, and by movement of the face, whether in speaking or chewing; in slight and early cases it may occur only under these influences. Thus a schoolmistress at first only suffered when speaking under excitement, *e. g.* to her class, and only after many months did it occur in quiet conversation. In another patient the spasm, always worst in the zygomatics, began in them, but only when reading aloud. When the influence of movement is great, the effect may be to cause the spasm very closely to resemble the "associated over-action" seen after facial paralysis. Frequently the spasm is increased by light and by cold. The influence of light is intelligible, since the orbicularis palpebrarum is almost always involved, and a strong light produces reflex contraction in this muscle under normal circumstances. Conversely the spasm is lessened by warmth and by darkness.

Facial spasm causes no pain; when pain is present it is independent and often primary. As a rule there is no paralysis. Voluntary movements may be interfered with by the spasm, but are otherwise unimpaired. Distinct loss of power only exists in cases of progressive organic lesions, especially in compression of the nerve, the effect of which is to cause first spasm, and then paralysis; or weakness and slight spasm may come on together.* Most, if not all, non-progress sive cases, in which considerable loss of power co-exists with spasm, are cases of old facial paralysis in which contracture and spasm have supervened. The electrical irritability of nerve and muscle is, as a rule, normal. I have found, however, in one case a distinct increase of irritability in the nerve.

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* As in the case recorded by Sharkey.

The spasm very rarely affects the muscles of the palate, even when the movement of the face is violent and general. In the case already mentioned, in which the spasm was due to the pressure of an aneurism on the nerve-trunk, the palate was unaffected. A few exceptions are on record. Thus, in a case of one-sided spasm, there were clonic contractions of the uvula, drawing it to the side affected, and synchronous with the spasm in the face (Schütz). In another case of bilateral spasm the uvula was drawn up by spasm of both sides, so that in the stronger contractions it almost disappeared (Leube). The palatine spasm is probably merely associated. I have seen bilateral contractions in the palate with unilateral facial spasm, but the two were not synchronous. In spasm of the two from pressure there has been compression also of the spinal accessory nerve (Oppenheim), and it may be noted that the palate and eyes have been affected together by nystagmus (*q. v.*).

Symptoms referable to spasm of the stapedius muscle have been very rarely noted, except in cases of tonic spasm of the orbicularis (blepharospasm). In these a continuous noise has been noted, and has persisted after the blepharospasm ceased.* Many persons can produce a quivering noise in the ear by a strong contraction of the orbiculares, and most readily if they try to turn the eyes up at the same time. In one case there was giddiness during the height of a paroxysm, with conjugate deviation of the eyes to the right, symptoms which Moos, who has recorded the case, attributes to spasm of the stapedius, suddenly lowering the pressure in the labyrinth and semicircular canals.

Subjective sensations of taste have not been noted, but taste was lost on the front of the tongue in one case, in which there was probably damage to the nerve. Equally rare is disturbance of the secretion of saliva, which was for a time excessive in the case of bilateral spasm in which the palate suffered. No other vaso-motor or trophic disturbance has been observed.

Occasionally the spasm spreads beyond the limit of the facial nerve, to the muscles of mastication, the tongue, and the muscles of the neck, and even of the arm.† It may begin in the tongue and spread to the face.‡ Extension to the eyeball muscles has not been hitherto recorded. In a case of my own there was slight deviation inwards of the eye on the side of the spasm during the paroxysms, apparently from spasm of the internal rectus or inhibition of the sixth nerve. In the case of Moös it is assumed that the deviation was secondary to derangement of the equilibrium centre by the labyrinthine disturbance.

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* Gottstein found that in such cases the sound could be arrested by pressure on the front of the mastoid process, or by electrical irritation of the skin at the same spot ('Archiv f. Ohrenheilkunde,' 1880, Bd. xvi, p. 61).

† Keen, 'Trans. American Surgical Association,' May 1st, 1886.

‡ Remak, 'Berl. kl. Wochenschr.,' 1883, No. 34.

Tender points in the fifth nerve are rare in general facial spasm, although they have been noted when the spasm was limited to the eyelids. Pain in the region of the fifth may occur at the onset, and has been already referred to. Occasionally there is some impaired sensibility of the face, due to greater damage to the fibres of this nerve.

Tonic spasm is common after paralysis, and exists in slight degree in the zygomatic muscles in some cases of old hemiplegia, and on both sides in paralysis agitans, in some cases of tetany, and in considerable degree in tetanus. Primary tonic spasm is said to have occasionally resulted from exposure to cold, to be unilateral or bilateral, and sometimes associated with trismus. Deep furrows are persistent; the mouth deviates towards the paralysed side; the palpebral fissure is narrower than on the other side, the eyebrow is raised, there is a sense of tension in the face, and movements are hindered.

Tonic spasm of the face may be produced in the cataleptic condition of hysteria. A curious form came under my notice in a man who, after sleeping for over eighteen hours, woke up in the following condition: there was some weakness of the left arm, none of the leg, no distinct paralysis of the tongue, and no paralysis of the face, but conspicuous over-action of the left side of the face, induced by the slightest excitement, coming on even when he entered a room, and passing away only after he had been perfectly quiet for a time. Sometimes it would occur without any exciting cause. The chief contracture was in the zygomatic muscles, causing a very deep naso-labial depression. There was also slight contracture of the orbicularis palpebrarum. He was readily excited to tears, and then the angle of the mouth was drawn still further outwards and the left eye almost closed. There was also a little rigidity of the muscles of mastication. The condition presented no change during five months that the patient was under observation. The features of the spasm that is secondary to torticollis, and of that which is allied to habit spasm, have been already mentioned.

Course.—Facial spasm usually begins gradually in the way described above, either by closure of the eyelids, or traction outwards of the angle of the mouth, and often many months pass before the other part of the side of the face is involved. The course of the affection is very irregular. Sometimes for weeks or months the face is comparatively or absolutely free from spasm, and then the movements return, spontaneously or after some emotion. The same proneness to relapse is seen when arrest has been obtained by treatment. The duration of the affection is always long; it generally lasts for years, and not unfrequently persists to the end of life.

PATHOLOGY.—The facts of organic disease show that the spasm may be due to a chronic irritative or "discharging" lesion in (1) the facial centre in the cortex, (2) the nucleus in the pons, or (3) the nerve-trunk. Since nerve-cells are the source of motor impulses, the two former are readily intelligible; whether cortical disease causes such spasm, or that which spreads as an epileptiform convulsion, must depend on a difference in the nature of the change in the cells, which is at present beyond our power of detection. It is less easy to understand how the third mechanism acts, although it has analogies in other parts of the nervous system. It may be by disturbing the downward nerve-currents from the facial nucleus, which are constant even during apparent rest. It is also conceivable that the pressure on the nerve may modify and derange the action of the facial nucleus itself; or, on the other hand, that the mechanical irritation of the nerve-fibres may excite them directly, as electrical stimulation does, and may intensify the impulses that descend them, continually or for the maintenance of tone, and for emotional and other expression. But we cannot thus explain the spontaneous paroxysms that characterise the affection. These must depend on a morbid state of the nucleus secondarily induced, either by an ascending neuritis or by the conduction of an influence in the opposite direction to that of habitual and chief function—such an influence as must descend sensory nerves when their irritation causes acute trophic disturbance. In the idiopathic form, we must assume a morbid action of the nerve-cells either in the nucleus or the cortex. Its persistent nature, even when its cause is transient, may be referred to the fact that functional action leaves a residual change disposing to its recurrence—an effect which we express as "lessened resistance" or "instability;" while, on the other hand, our means of counteracting this tendency are extremely limited.

When facial spasm is due to a reflex cause, as neuralgia or exposure to cold, it is reasonable to regard the nucleus as the part chiefly affected. In the cases that are excited by depressing emotion, or develop slowly without apparent cause, there is nothing to show whether the deranged action is at the cortex or at the nucleus. In either position the extension of the spasm to the other side is readily intelligible, much more so than its limitation to muscles on one side (as the orbiculares palpebrarum) which habitually act with their fellows; and in either case, the very different associations of the orbicularis oris enable us to understand its escape.

DIAGNOSIS.—The chief point in diagnosis is the distinction of primary spasm from that which follows paralysis, and this rests on the history or the presence of weakness, which may always be observed on energetic movement, and on the existence of persistent contracture and over-action, as well as of occasional spasm. In rare cases of chorea the face is much affected, and the limbs but little; and it is probable that such a case has been mistaken for, and even published as, a case of simple facial spasm. Attention to the condition of the limbs will prevent error, since slight movements may always be observed in them. In hysteria there is either tonic contracture, especially in the orbicularis, or attacks of quivering movement, which do not resemble true facial spasm.

A more urgent question is the seat and nature of the disease. In forming an opinion on this point we must be guided by the considerations already mentioned in the sections on causation and pathology. More facts are needed regarding the relation between the character of the spasm and the seat of the disease. Partial facial spasm, however, *e. g.* limited to the zygomatics, is more likely to be cortical than that which involves the greater part of one side of the face. Any tender points, or source of irritation in the fifth nerve, or a history of pain, suggest a reflex origin and nuclear seat. Indications of increasing weakness, secondary in time, suggest a progressive organic cause.

PROGNOSIS.—The prognosis is grave in all cases except those that are of recent origin, and are distinctly due to a reflex cause; and even in these the cessation of the spasm cannot be certainly expected to follow the removal of the cause. In cases which have lasted more than a few months the probability is against recovery, although the fact that in a few instances, even of years' duration, recovery has taken place, justifies an effort to relieve. Only when the symptom is due to progressive organic disease has it any intrinsic gravity. But, trivial as it is in other cases, it causes more annoyance than many diseases of far more serious nature.

TREATMENT.—In a recent case, apparently excited by cold, free diaphoresis should be employed, and the face and side of the head bathed frequently with hot water. If there are indications of organic disease, the nature of this must be ascertained, and, as far as possible, treated. All causes of reflex irritation must be sought for and removed; decayed teeth should be extracted, especially if they are on the same side as the spasm. Any derangement of the general health should also be treated, and in the cases that are excited by depressing emotion, tonics are usually required. So-called nervine tonics and stimulants, zinc, nitrate of silver, asafœtida, valerian, have all been recommended, but in the vast majority of cases they conspicuously fail. In only one typical case have I known recovery to occur under such treatment, which consisted of arsenic, bromide, and Indian hemp. Hypodermic injections of strychnia are said to have cured one case (Sanders).

It is chiefly by the administration of sedatives that attempts have been made to reduce the activity of the cells and lessen the spasm. Almost every drug of the kind has been employed, and some have been said to be successful in isolated cases, such as gelsemium, conium, and morphia. The last, given under the skin, has most frequently done good, either alone or combined with atropine. From the results obtained in torticollis, it is probable that a permanent effect may be produced by keeping up the influence of morphia for many months; but this course of treatment may easily have results more serious than the malady itself.

Electricity has been largely used, and has been highly praised by some of its advocates, but in most cases it fails even to relieve. Only a weak voltaic current should be employed, in a "stabile," *i. e.* uninterrupted manner. Various methods of application have been recommended, and may be tried. One is to place the anode in front of the ear, the kathode on the muscle, so that a descending current may pass. Another is to place the anode on the nerve, or behind the ear, or at the occiput, the kathode on some indifferent place, so as to obtain the simple sedative influence of the positive pole. Another is to place a pole on each mastoid process.

The diminution of the sensitiveness of the fifth nerve by cocaine seems useless. Indeed, it is important to remember that strong sensory impulses from the seat of the spasm have more power than any other agency to raise local resistance and arrest over-action. This opposition of reflex influences is a familiar fact (see vol. i, p. 200). We see an instance of the mechanism in the effect of a blister on convulsions that begin locally, but the effect is transient, and seldom obtained in persistent spasm outside the realm of hysteria. I have, however, once known facial spasm of two months' duration to cease permanently on an attack of neuralgia in the face. Similarly, the spasm has been observed to cease during the existence of a painful affection of the conjunctiva (Bernhardt). Counter-irritation, as by a blister behind the ear, generally causes some diminution of the spasm for a time, perhaps by the inhibitory influence of the sensory irritation, but no lasting amelioration results. The application of the actual cautery along the cervical spine is said to have cured one case (Remak).

Stretching of the facial nerve has been of late adopted in these cases, but unfortunately with imperfect realisation of the hopes that were entertained when the operation was introduced, and that seemed to be justified by its immediate effect. Slight stretching of the nerve seldom has any influence.* Vigorous stretching causes paralysis of the face, lasting for weeks or months, with degenerative reaction in the nerve and muscle. The paralysis in time passes away. In many cases the spasm has returned with voluntary power. In others the face has remained free from spasm for some time after the recovery of the nerve, but the spasm has ultimately returned, either spontaneously, or on some fresh exciting cause, such as an emotional shock. Of thirteen cases collected by Godlee,* in only one (recorded by Southam†) was the patient free from spasm two years after the operation; and in a more recent collection of twenty cases by Keen, the only other case of cessation for a longer period than six months is that of Zeiss, already mentioned.‡ In some cases the spasm became as severe as ever, in others it was distinctly less intense than before the operation and remained so. The operation should probably be performed only in the cases in which there is a possibility that the source of the spasm is disordered function of the facial nucleus, and it would be desirable not to trust to the operation alone, but to follow it up by a course of some other treatment, such as the hypodermic injection of arsenic and morphia, which is known to have some power of lessening such spasm; this should be commenced when the palsy is passing away. The operation may perhaps have an upward influence on the nerve-cells of the nucleus, as well as that due to the counter-irritation it effects through the fifth nerve. The unfortunate tendency to recurrence after some months, which most cases present, may perhaps be due to the same influences that lead to secondary spasm after idiopathic neuritis, and which we are still unable to counteract.

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* In a case recorded by Zeiss ('Wien. med. Wochenschr.,' 1884, No. 2, and 1885, No. 27), stretching insufficient to cause paralysis was followed by slow diminution in a spasm of eight years' duration, which disappeared seven weeks after the operation, and was still absent two and a half years later.

BLEPHAROSPASM.—Spasm confined to the eyelids, and showing no signs of extension to other parts of the face, is distinguished from other forms of facial spasm also by its causes and its bilateral character. It occurs in *tonic* form in connection with photophobia, as a result of painful affections of the eye, and sometimes of other branches of the fifth nerve. It is an excessive action of the reflex mechanism that normally guards the eye. When thus set up it may persist as a troublesome affection in excess of its cause, and may continue in the dark, and even exist in an eye that is blind. *Clonic* spasm of the eyelids is sometimes dignified by the name "nictitation," and sometimes occurs in hysteria, and also in children as part of "habit-chorea," which is elsewhere described. The most important elements in treatment are the removal of nerve irritation, for which in the case of conjunctival disease cocaine is useful, and the diminution of the central over-action by sedatives, such as bromide and belladonna, and by tonics such as iron and quinine. Cold douches to the eye should also be used.

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* 'Trans. Clin. Soc.,' vol. xvi, p. 220. In a previous paper by the same author (ib., vol. xiv, p. 44) the method of performing the operation is fully described. It is also described by Keen, loc. cit. (p. 233).

† 'Lancet,' August 27th, 1881, and a later note in Godlee's table.

‡ In the case of a man of fifty, under my care, in whom Mr. Godlee stretched the nerve, the contractions have now been absent for a year and three quarters. The onset was after exposure to malaria, but the patient has, for a year, returned to the same conditions. The spasm was characteristic, left-sided, but the case was unusual in that there was slightly lessened sensibility in the second division of the fifth nerve.

AUDITORY NERVE; DISTURBANCE OF HEARING.

It is probable that the fibres of the auditory nerve that go to the cochlea on the one hand, and those that supply the semicircular canals on the other, differ in function, and that only the former are excited by sound. The latter seem to convey impressions from the canals, generated by the pressure or movement of the endolymph in various postures and movements of the body. Hence the latter fibres have been distinguished by Cyon as the "space-nerve" (*Raumnerv*), and, although the term is awkward, a better one has not hitherto been suggested. The two sets of fibres unite in a common trunk, in which they cannot be distinguished. Within the internal auditory meatus and at the base of the brain the auditory nerve is adjacent to the facial. The deep origin of the nerve has been already described (p. 47), and we have seen that many fibres pass by the inferior peduncle to the cerebellum, with which also the nuclei are probably connected. The former probably convey impressions from the canals to the equilibrial mechanism in the middle lobe. The upward auditory path seems to pass by the superior fibres of the tegmentum of the crus* to the internal capsule, but whether it reaches the tegmentum through the pons or through the cerebellum has not yet been ascertained. There is certainly a decussation above (or at the level of) the nuclei, so that the auditory path from each ear passes to the opposite hemisphere, and apparently in or near the posterior extremity of the internal capsule. The cortical centre for hearing is seated in the first temporo-sphenoidal convolution (see p. 24), and that of the left side subserves the auditory use of words.

Disturbance of the functions of the auditory nerve are much more often due to disease of the ear than to affections of the nerve-trunk or centres. Our knowledge of the symptoms that are of primary nerve origin is still very imperfect, because they are for the most part identical with those due to lesions of the labyrinth. Exact aural diagnosis stops at the middle ear; beyond this it resembles that of affections of the eye before the invention of the ophthalmoscope. Labyrinthine and nerve lesions are now confused under the term "nervous deafness," just as affections of the retina, choroid, and optic nerve were included under the common term "amaurosis." Indeed, since labyrinthine lesions cause symptoms by their influence on the nerve-fibres, the confusion seems almost inevitable, unless associated symptoms point definitely to a given seat of disease.

Five kinds of symptoms may result from disease of the auditory nerve in its course or termination. (1) Loss of function: deafness. (2) Increased action: auditory hyperæsthesia, "hyperacusis." (3) Irritation symptoms in the auditory function of the nerve, causing subjective sensations of sound: "tinnitus aurium." (4) Disturbance of equilibrium, or sensations of such disturbance, due to derangement of the fibres from the semicircular canals. (5) Connected with the last (but too rare to merit more than a passing mention) are certain involuntary movements that have been observed in cases of disease of the nerve within the ear. Pendulum-like oscillations of the head were present in two cases recorded by Moos.* The fourth class of symptoms, which constitute "labyrinthine" or "auditory vertigo," can be considered more conveniently in connection with other forms of vertigo. Here only the derangements of the auditory function of the nerve can be briefly considered in their medical relations. For a fuller description of the symptoms of labyrinthine affections the reader is referred to special works on Diseases of the Ear.

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* See p. 49.

Diminished Function; Nervous Deafness.

Causes.—Deafness may be congenital, as in 80 per cent. of deaf mutes; in the remaining 20 per cent. it is the result of disease early in life. The mechanism of congenital deafness is not known. Inheritance is a potent cause, especially that which results from the marriage of relations in families in which the defect exists; rarely intermarriage apart from the defect gives rise to it. As an instance of inheritance, one family in the United States has had thirty-four cases in four generations, another twenty-one in three. This condition, however, does not come within the subject as here considered.† The causes of "nerve-deafness" may be thus enumerated.

(1) By far the most common is disease of the labyrinth, either hindering the passage of the vibrations to the structures in which the nerve-fibres end, or else damaging those structures and the nerve-endings themselves. Such disease may be primary in the labyrinth, or may extend to it from the middle ear. Our knowledge of the nature of the morbid processes is limited to the occurrence of acute inflammation, chronic inflammation, syphilitic disease,‡ degenerative processes, and probably hæmorrhage. From these various tissue changes, fibrous and calcareous formation may result. Primary inflammation is sometimes bilateral, and thus causes deafness on each side; it may result from inherited syphilis, cold, and some toxic blood-states. It occurs also in purulent meningitis by extension along the aqueduct of the cochlea and along the auditory nerve itself, by its substance or outer layer. In such cases the labyrinth, often on both sides, contains pus, and also the organisms found in the mem-

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* 'Zeitsch. f. Ohrenheilk.,' xii, p. 101.

† For a summary of facts see Warden, 'Brit. Med. Journ.,' 1887.

‡ Moos, 'Virchow's Archiv,' Bd. lxix, p. 313 (with autopsy); see also McBride, 'Glasgow Med. Journal,' 1885, p. 172.

branes in cerebro-spinal meningitis and in the tubercular form.* It is probable that the membrane of the labyrinth, in the old, undergoes degenerative changes, since these may be observed in the membrana tympani. In those who are gouty, symptoms sometimes suggest that the fibrous tissues of the internal ear may be the seat of changes similar to those met with elsewhere in this condition.

Certain drugs cause deafness having the characters of labyrinthine deafness, and probably so because congestion of the internal ear has been found to accompany the symptoms. A loud noise has been known to cause permanent deafness. Lucae suggests that the mechanism may be a hæmorrhage into the labyrinth, since he has found an extravasation in the middle ear from this cause.†

(2) Much less common than disease within the internal ear are lesions of the nerve-trunk in the internal auditory meatus or at the base of the brain. The nerve suffers chiefly from disease commencing outside it, especially morbid growths or meningitis, of syphilitic or other nature, and aneurisms. Inflammation readily invades it, on account of the slight protection afforded by its sheath. It may also be compressed in consequence of thickening of the cranial bones narrowing the meatus. Disease arising in the nerve itself is rare; tumours (neuromata), interstitial hæmorrhage, and calcareous nodules have been met with. Primary inflammation has not been proved to occur, although an auditory neuritis analogous to optic neuritis has been suspected in cases of cerebral tumour. In cases described as rheumatic neuritis an affection of the labyrinth is more probable.

Primary degeneration of the nerve occurs occasionally in locomotor ataxy (Erb, Wernicke). A case is mentioned in vol. i in which the progressive limitation of the range of hearing made such atrophy of the auditory nerve highly probable, but this morbid process has certainly been sometimes assumed to exist without sufficient grounds, and the symptoms of simple "nervous deafness" in tabes are no proof of a primary atrophy. Such degeneration has also been met with as an isolated change, chiefly in the old, as in a woman eighty-nine years of age in which this atrophy was found by Lucae. Concretions of phosphate of lime have also been observed in the substance of the nerve (Moos). Long-continued disease of the nerve, in consequence of disease of the middle ear, has been supposed to lead to atrophy, but the evidence is inconclusive.

(3) The nuclei within the pons may be damaged by hæmorrhage, softening, or tumour, but seem to escape more frequently than other nuclei.

(4) Deafness is sometimes produced by disease above the nuclei. It may be caused (*a*) by disease which damages the superficial layer

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* See a series of papers by Steinbrugge, 'Zeitschrift f. Ohrenh.,' &c., 1886–7–8, and Gradenigo, 'Ann. de Mal. de l'Or.,' 1890.

† Lucae, 'Subjectiv. Gehörs-empfindungen,' 1884.

of the tegmentum, such as a tumour of the corpora quadrigemina;[*] (b) by disease of the internal capsule; it is then associated with hemianæsthesia and impairment of the other special senses; (c) by disease of the cortex in the upper part of the opposite temporo-sphenoidal lobe, or the white substance within it.[†]

(5) Loss of hearing may be of functional origin. In hysteria it is common as part of hemianæsthesia. In anæmia there may be some loss of hearing, which may pass away when the general health is improved. Considerable loss of blood has been followed by absolute deafness, analogous perhaps to the blindness that results from the same cause; after death no morbid condition of ear or nerve has been discovered.

(6) Lastly, slight persistent defect, having the characters of "nerve-deafness," is sometimes congenital, and may occur in several members of a family without traceable cause. Some defect of articulation may accompany it, and occasionally general mental backwardness. The condition probably depends on a congenital local defect of the brain, and may be regarded as allied to that of congenital deaf-mutism, which is apparently analogous in nature but more severe in degree.

Symptoms.—Deafness, the indication of lessened function of the auditory nerves, is due to so many morbid states of the ear, that alone it is of little value as an indication of an affection of the nerve. For a full description of the methods of examination of the ear the reader is referred to special works.[‡] Some points of medical importance may be here mentioned. When the deafness is due to obstruction of the meatus, or to disease of the middle ear, *i. e.* whenever there is impaired conduction of vibrations to the internal ear, and no disease of the labyrinth itself, there is deafness to vibrations that reach the ear through the air, while those that are conducted through the bones of the skull can still be perceived. A vibrating tuning-fork held opposite the meatus is inaudible, but it is heard at once if placed in contact with the bone of the skull. In cases of slighter deafness, in which the tuning-fork can be heard through the air, defect of conduction can be demonstrated in another way. Normally, as the vibrations lessen, they can be heard through the air longer than through the bone; when the fork in contact with the skull ceases to be heard, the sound can still be perceived if the instrument is held opposite the external meatus ("Rinne's test"). This is because the receptive mechanism is more sensitive to vibrations that reach it through the tympanum and chain of bones than to those which it receives from the bone directly. If the tuning-fork cannot be heard longer through the air than through the bone, there is impaired conduction through the meatus or middle ear.[*]

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[*] See chapter on "Localisation."

[†] Deafness in the left ear, with left-sided loss of sensation, was produced by a tumour of the right hemisphere in a case recorded by Strümpell, 'Neur. Centralbl.,' 1882. See also Sharkey's case, fig. 15, p. 23. In a man of seventy-nine left-sided deafness, after a fall, was found to be due to occlusion of the right posterior Sylvian artery, with softening of the region adjacent to the posterior limb of the fissure of Sylvius, the ear being normal (Kauffmann, 1886).

[‡] An examination of the external meatus should, of course, never be omitted, and I may, in passing, mention the very great suitability of the ophthalmoscope for this purpose. A three-inch lens should be held over the speculum, and be slightly tilted to get rid of the reflection. The lens concentrates the light on, and at the same time magnifies, the membrana tympani. A light vulcanite speculum answers best.

Sounds conducted through the bone are intensified by closure of the external meatus. This is due to the circumstances that vibrations escape by the open meatus[†] (Mach), and that the closure of the meatus intensifies the vibration by converting the cavity into a resonant chamber (Lucae). The intensification shows that hearing through the bone is normal, *i. e.* that the labyrinth is sensitive. But it is a less convenient test than simple contact, because unobservant persons often fail to recognise the increase. If there is some deafness to sounds through the air, and yet the tuning-fork is heard longer through the air than through the bone, *i. e.* the hearing through air and bone have their normal relation, the deafness is due to the nerve or labyrinth.

Nerve-deafness is usually greatest for sounds of high pitch and short duration. A valuable instrument for testing the audibility of high-pitched sounds is Galton's whistle, the notes of which reach the limitation of hearing and extend beyond it.[‡] Deafness to all its sounds is unquestionably pathological, and inability to hear the higher notes on one side only has the same significance.

The high-pitched short sound of a watch furnishes a test of considerable value and delicacy for the ability to hear through the bone, but caution is needed in drawing inferences from the results obtained. The best method of using it is to close the meatus by pressing back the antitragus, and first to hold the watch near but not in contact with the root of the zygoma, and then to press it firmly against the bone. The observation may be repeated against the mastoid process. If the watch is heard better in contact than when not in contact, the function of the labyrinth is not impaired. If it is not heard better in contact, further observations may be made with the tuning-fork. If the watch is heard through the bone, it is not likely that the tuning-fork will reveal impairment. If the sound is heard through the bone but not so loudly as normal, we cannot infer disease of the labyrinth or nerve, because simple anchylosis of the stapes will reduce bone conduction, although no disease of the middle ear will extinguish it (see note on last page). But during the degenerative period of life, after fifty or sixty years of age, it is very common for the watch not to be heard through the bone, although there is no other evidence of impaired function. The loss apparently depends on the labyrinthine changes incidental to age already mentioned. During the first half of life it is rare to find bone-deafness, except as a pathological condition, and this is true at any age if the defect exists on one side only. Even after middle life a bilateral loss is of significance if associated with deafness to Galton's whistle through the air.

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[*] There is reason to believe that some of the vibrations reach the labyrinth from the bone through the tympanum; but that vibrations may pass to the labyrinth directly, seems proved by the fact (observed by Lucae) that when the membrane and bones of the tympanum are congenitally absent, sounds can still be heard through the bone.

[†] If a stethoscope or otoscope is applied to one meatus, sounds entering the other ear can be distinctly heard; the old adage, "in at one ear and out at the other," having thus a basis of literal truth.

[‡] A cat starts up when a note is (presumably) sounded which is quite inaudible to man.

The state of hearing through the bone is of special importance to the physician, because it indicates the functional state of the internal ear and nerve, and eliminates the morbid states of the conducting media, the meatus and tympanic cavity. It is assumed that the vibrations pass from the bone directly to the labyrinth (see note on preceding page).

Another indication of impaired function of the nerve is an altered electrical reaction, first ascertained by Brenner, and since studied by Erb, Gradenigo, and others. But this is little employed in diagnosis. The attempt to obtain it often causes vertigo; a current of moderate strength (10—16 milliampères) gives a reaction only in a small percentage of normal individuals, and the order of response is variable; moreover an alteration occurs in morbid states of the middle ear as well as in affections of the nerve, and in the latter is sometimes absent.

We have no means of distinguishing between disease of the labyrinth and nerve except by the associated symptoms. In each case the deafness is the same, and is often associated with symptoms of irritation—subjective sounds. If the facial nerve is paralysed, and there is no disease of the middle ear or bone, we may feel sure that the nerves are affected at the base of the brain or in the internal meatus.* A basal affection is also probable if any other nerve near the auditory (as the sixth) suffers with it. In some instances the auditory nerve is damaged without the facial at the base of the brain because the resisting power of the *portio mollis* is less than that of the *portio dura*. This is especially the case in meningitis, in which the facial nerve has been found but little damaged, while the auditory was softened and infiltrated with the products of inflammation and with extravasated blood (Gradenigo).

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* If, with this combination of symptoms, there is also disease of the middle ear, it is probable that the facial nerve and the labyrinth are affected by extension of disease from the tympanum, but this diagnosis is not certain, as a case recorded by Schwartze shows. In spite of disease of the middle ear, the deafness and facial paralysis were found to depend on a tubercular tumour springing from the dura mater, and compressing the trunks of the facial and auditory nerve ('Beiträge zur Path. des Ohres,' 1870).

The symptoms of disease of the auditory nuclei in the medulla are still little known. In several recorded cases, disease of the nuclei on one side has caused deafness in the ear on the same side. But we do not know whether this relation is invariable, whether unilateral disease always causes deafness on the same side, or whether the connection of nucleus and ear is in part crossed.* Moreover, extensive damage to the nuclei has been found when no deafness was noted. Nuclear disease may be suspected if deafness comes on suddenly, together with other symptoms of a lesion of the pons or medulla, especially with weakness of the limbs on the opposite side. Sudden deafness, alone, is of no localising significance, because either the trunk of the nerve or the labyrinth may be the seat of sudden hæmorrhage. Deafness of gradual onset, in association with bulbar symptoms, is also of little significance as regards disease of the nucleus, since it is more frequently due to pressure on the nerve. Deafness, with hemianæsthesia or hemiplegia on the same side, is probably due to disease of the cerebral hemisphere.

Bilateral deafness may be due to various causes:—(1) Symmetrical disease of the labyrinth, which is common; acute inflammation is sometimes, and chronic degeneration is often, bilateral. Double otitis interna has sometimes been mistaken for meningitis, especially in the young, in whom it is attended with fever, and sometimes with general cerebral symptoms and even convulsion. It may cause unsteadiness of movement, but this is usually transient from bilateral disease. (2) Symmetrical lesions of the two auditory nerves, which are very rare. (3) Possibly disease of the medulla. (4) A tumour in the corpora quadrigemina, damaging the crustæ of the crura cerebri. (5) Symmetrical disease of each temporo-sphenoidal lobe; syphilitic gummata caused this effect in a case recorded by Wernicke and Friedländer.

The pathological changes in disease of the labyrinth are outside our province. Those of disease of the nerve-trunk are multiform and variable, depending on the nature of the primary lesion. Evidence of inflammation, differing in extent and degree, is present in acute meningitis, and at a later stage, or in chronic processes, degeneration of the fibres is found. It deserves note, however, that when these have been destroyed for many years (*e. g.* by meningitis) the cells of the auditory nuclei have been found normal.†

Treatment.—For a description of the little that can be done in the treatment of disease of the labyrinth the reader is referred to special treatises. The treatment of disease of the nerve-trunk or centres is for the most part that of the morbid process, and must be conducted on the same principles as that of similar lesions in the other cranial nerves. In cases of acute deafness, counter-irritation, as by blisters, is often of great service, and even in chronic cases may do some good. The auditory nerve may be stimulated by electricity, but the results obtained by most of those who have employed this method have been slight and disappointing.

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* See a curious case of tumour of the right cerebellar amygdala (Wolf, 'Archiv f. Ohrenheilk.,' Bd. xvi, p. 157) which compressed the right auditory region, and is said to have caused deafness of the left ear and left facial paralysis. The latter, however, might have been due to a tumour that existed in the ascending parietal convolution.

† Schultze, 'Virchow's Archiv,' Bd. cxix, 1890.

AUDITORY HYPERÆSTHESIA.

The term is applied to an increased or perverted action of the auditory nerve or centres. While deafness is much more frequently due to diseases of the ear than to morbid states of the nerve, the opposite is true of hyperæsthesia, which is commonly due to altered nerve-function.

(1) *True hyperæsthesia*, increased keenness of hearing, "hyperacusis," is a rare morbid state in which sounds are heard with undue loudness, and even such as are inaudible to other persons are distinctly perceived. It occurs chiefly in hysteria, usually in association with augmented acuteness of other senses, and is probably, in such cases, of central origin. It has also been observed at the onset of acute cerebral and general diseases, and may co-exist with tinnitus as a consequence of the same cause. The changes that give rise to spontaneous sounds may increase the intensity of those that are heard, as cutaneous hyperæsthesia occurs in neuralgia and superficial tabetic pain. The hyperacusis may be very marked for the high notes of Galton's whistle.

From true hyperæsthesia we must distinguish certain forms of hyperacusis due to disorder of the conducting mechanism. In paralysis of the stapedius muscle, from disease of the facial nerve, low notes may be heard with undue loudness.

(2) *Auditory Dysæsthesia*, "*Dysacusis*."—Sounds, although not heard with undue loudness, cause discomfort. This is common in cerebral affections, functional and organic, during attacks of headache, in many cases of meningitis, &c. The pain in the head seems to be intensified by the noise. There is no evidence that irritation of the auditory fibres ever produces pain directly. Politzer has indeed recorded a case of labyrinthine disease in which chords played on an harmonium caused a distinct sensation of pain, while single notes did not; but the case stands alone, and it is probable that the pain was produced in some indirect manner.

The treatment of these forms of auditory over-action is that of the morbid state with which they are connected, and the nature of this is usually obvious. If direct treatment is needed, full doses of bromide of potassium have most influence in diminishing the morbid excitability.

IRRITATION OF THE AUDITORY NERVE; TINNITUS AURIUM.

Subjective sounds are of varied character and still more varied origin. They constitute a common ailment, and one that is often most distressing and intractable, which disturbs the whole nervous system, and thus tends to become worse. Its frequency is not surprising. When we consider, on the one hand, how exquisitely sensitive the organ of hearing is, how slight a vibration will affect it, and, on the other hand, the facts that around, and even within it, blood is in constant and muscle in frequent motion, and that all parts of it are liable to disease of various nature, the marvel is rather that silence is habitual. It has indeed been thought, with reason, that the apparent silence is due to cerebral inattention, and not to peripheral inactivity. In a quiet room, continuous auditory sensations may be noticed, of which we are normally unaware, just as in the dark the field of vision may be found full of moving points of light, or sensations may be perceived from any part of the body.

Almost any morbid process in any part of the ear may cause subjective sounds; they may result also from sound-producing processes in and about a normal ear, as well as from irritation of the auditory nerve and centres within the skull. But of all the causes, changes in the labyrinth are by far the most common; evidence of "nerve-deafness," mostly due to changes in the internal ear, are distinct in four fifths of the cases which come under the physician's notice. The experience of aural surgeons seems to furnish a large proportion of cases of disease of the middle ear, but for this there may be special reasons. In a few cases the symptoms suggest that the morbid process is wholly in the auditory centres, the nucleus of the nerve or cortical structures.

The chief predisposing causes are—(1) those that lead to labyrinthine disease, among which gout is especially frequent; and (2) a neurotic disposition, which probably acts by rendering the nerve-elements prone to generate impulses when diseased, and by disposing the centres to intensify these impulses, and even to produce them in some cases of central origin. Many sufferers have been liable to neuralgia (with which tinnitus presents analogies), to periodical headaches, to typical migraine, or to cephalic sensations of various character. The symptom is very rare in the young; in most sufferers it begins after middle life, and it is common in the old.

The more direct causes may be thus enumerated:—(1) The blood-currents within and near the ear, which normally give rise to no sound, may be so changed that they cease to be noiseless. In anæmia, vibrations occur in the blood with undue readiness, and a pulsating murmur may be heard, probably produced in the carotid artery. A similar murmur may also arise in an intra-cranial aneurism. Vaso-motor paralysis of the labyrinthine vessels has probably been the cause of a subjective sound observed in a case of disease of the cervical spine, pressing on the vertebral artery along which the sympathetic fibres run. A similar mechanism may have been effective in another case in which there was enlargement of the glands of the neck adjacent to the sympathetic; the noise ceased when the glands became small.* Perhaps dilatation of the small arteries within the ear may give rise to a similar sound, and if tinnitus is ever, as is asserted, of reflex origin, it may be thus produced. Pulsating sounds are, as a rule, arrested or lessened by compression of the carotid artery in the neck.

(2) Tinnitus may attend every form of ear disease—accumulation of cerumen or growths in the external meatus; inflammation of the middle ear, catarrhal, plastic, or suppurative; spasmodic contractions of the tympanic muscles, and various morbid states of the labyrinth. The precise mechanism by which the sounds are produced is a matter of conjecture; vascular congestion, increased labyrinthine pressure, and irritation of the nerve-endings or the structures connected with them, have all been assumed, and may all be operative in different cases.

(3) Organic changes that irritate the fibres of the auditory nerve or its centres may cause subjective sensations of sound, and these may therefore be present as a symptom of any of the morbid states already mentioned as affecting these parts, and producing loss of hearing, degeneration, concretions, &c.

(4) Lasting tinnitus sometimes follows a fall in which the head is struck. In some cases there are signs of labyrinthine deafness; occasionally there is hyperacusis and no deafness.

(5) Intense stimulation of the auditory nerve, as by a loud railway whistle, has been known to set up a lasting subjective sound, but by what mechanism is unknown. Habitual exposure to sounds may cause tinnitus, usually slight in degree; musicians, piano tuners, smiths, &c., are said to suffer occasionally in this way.

(6) Subjective sounds are certainly sometimes due to a functional disturbance of the auditory centres, and probably have this origin in cases in which we cannot feel sure of the fact. In migraine, paroxysmal sounds sometimes occur, although they are a rare symptom. Such sounds are more common as the aura of an epileptic seizure; it is probable that they are due to disturbance in the cortical auditory centre, since organic disease in this position may cause such a sound as the warning of a convulsion.† That continuous tinnitus sometimes results from central functional disturbance is suggested by the elaborate character of the sounds. In various conditions of nervous exhaustion and excitability, tinnitus may arise without impairment of hearing. Of the pathology of these cases nothing is certainly known, but it is probable that they are due to defective nutrition of the nerve-elements, central or peripheral.

It must be remembered that tinnitus is perceived in consequence of the activity of the cortical nerve-cells, and that in labyrinthine disease there must be an intermediate excitation of the auditory nuclei. The susceptibility of the nerve-cells will be increased by whatever impairs the general nutrition of the nervous system, and hence the effect of ill-health, mental disturbance, and the like, in making the sounds louder, can be understood. Moreover, the symptoms of many cases suggest that, even when tinnitus is ultimately due to changes in the ear, it may be increased, maintained, and modified in character by derangement of the function of the auditory centres. Their continuous activity must entail secondary changes in their nutrition and function, which may well proceed to independent co-operation, especially in those predisposed to disturbance of the nervous system. On this hypothesis many facts, otherwise obscure, become clear.

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* These two cases are recorded by Brandeis, 'Zeitsch. f. Ohrenheilk.,' xi, 1882, p. 294.

† See p. 24; also "Localisation."

SYMPTOMS.—The sounds may be referred to one or both ears, or to the head, either behind or above the ear, the occiput or the vertex. This is sometimes called "tinnitus capitis." The sounds are extremely various in character and intensity. Slight sounds may be low-pitched, a low rumble like a distant waggon, or a faint murmur such as may be heard when a shell is held up to the ear. Louder sounds are rarely low in tone. They may be humming, hissing, rushing, or roaring noises. A common simile is that of an engine or threshing machine, or the hissing of a kettle or a gas jet, while still louder noises are compared to that of a steam-engine close at hand, or a waterfall, or to a room full of machinery in motion. Others again, less common, are like the sound of a bell, or of many bells jangling at a distance, while still more elaborate sensations may resemble music or voices. Definite words are seldom heard, except in cases of insanity or epilepsy.

It is common for sounds to vary at different times. In a case of bilateral nerve-deafness, a continuous loud rushing sound, referred to the head, would sometimes stop for a few minutes, and begin again as a whistle, to pass presently into its usual form. With the same condition, another patient described four sounds, hissing, whistling, &c., which were sometimes successive and sometimes seemed all to be heard at once.

An important difference in the more simple sounds is their continuity. Some are rhythmical, and then are usually synchronous with the pulse; others are continuous and unvarying. The significance of the synchronism is that the sound is due, directly or indirectly, to the influence of arterial pulsation, but its ultimate cause may be either an increase of this pulsation or an increased sensitiveness of the nerve-structures. Hence the sound is intermitting, not only in aneurism, but in anæmia, and also in many diseases of the middle ear, and in some labyrinthine affections, and even occasionally in morbid states of the centre.* Sounds of central origin are generally continuous; but continuous sounds are also often due to ear disease, labyrinthine or tympanic, and even to accumulations of wax in the meatus. In one case of slight bilateral labyrinthine deafness, a continuous sound, referred to the vertex (sometimes like distant bells) was accompanied, when the patient was tired, by a pulsating sound in one ear. In another, of similar character, the sound, also referred to the head, was sometimes a continuous hiss, sometimes a pulsating noise. Thus this feature of tinnitus affords little help in the diagnosis of its cause. Elaborate sounds are generally of central origin. A lady, for twenty years, has heard the sound of music; no actual tune can ever be distinguished, but she states that were it not for its persistence the sound would be pleasant: no affection of the ear can be discovered. Auditory hallucinations of the insane, although of cerebral origin, are sometimes determined by the presence of ordinary tinnitus, which existed before the onset of the hallucinations. The sounds that are heard in epilepsy may be simple or elaborate, or both. Thus, before severe and during minor attacks, one patient heard a noise like the sea waves breaking, and with this a distinct sound of the voices of friends; as the attack was passing off she always seemed to hear her husband telling her not to be alarmed, even when he was not present. The aura of tinnitus may also be accompanied by complete word-deafness, a complex condition combining inhibition and discharge (see "Epilepsy").

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* That intermittent sounds may be of central origin is certain, from the facts of

At the beginning of the affection, the sound, especially if slight and of a familiar character, may be thought to have an actual objective origin. This is especially the case when the sound is elaborate. One patient sent a message to his next door neighbour, asking that a clock might be moved, the loud striking of which annoyed him; he had no idea that the sound was subjective until informed that there was not a striking clock in his neighbour's house. In most cases, however, the real nature of the sound is quickly recognised. The sound is usually referred by the patient to the ear; when it seems to be in the head generally, the difference depends in part on the loudness of the sound and partly on its bilateral character, on its source, and also on conditions we do not yet understand. The influence of intensity was shown by one patient who said that the sound, as a rule, seemed to be in the ears, but when it became more intense it seemed to be within the skull. The effect of the bilateral character of the sound on its apparent seat agrees with the fact that, under normal circumstances, a sound conveyed simultaneously to both ears by a double tube is referred to some region in the middle line of the skull, the precise locality varying in different persons. But sounds are not seldom referred to the head when there is nerve-deafness on

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epilepsy. I have elsewhere recorded a case in which part of the aura was an intermittent hissing, evidently synchronous with the pulse, and succeeded by two bright lights, which seemed to approach the patient by jerks, of the same rhythm as the preceding sounds ('Epilepsy, &c.,' p. 67).

one side only, sometimes to some part of the hinder half of the head on that side, rarely to the vertex.

Why sounds should ever be referred to the head may seem a difficult problem, since in all normal stimulation of the auditory nerve the cause of this is referred to an external source. The solution of the difficulty is perhaps to be found in two facts. (1) Vibrations that reach the labyrinth by the bones of the skull are referred to the bone; this agrees with the fact (see p. 264) that the vibrations reach the labyrinth directly, and must influence the nerve-endings in a special manner, differing from that produced by vibrations that pass through the chain of bones in the middle ear. It is easy to understand that, in labyrinthine disease, the irregular irritation of the nerve-endings should correspond to the former rather than to the latter form of excitation, and that a similar irregular excitation should occur in the cells of the cerebral structures in some cases of tinnitus produced in them. (2) Many of the sounds conducted through the middle ear are elaborate in character, and when such subjective sounds occur they are usually referred to a source external to the body, even though they are due to central disturbance.

The sounds may vary from time to time in their intensity. Such variations may be distinctly dependent on the general health of the patient; the sounds may lessen or even cease during good health, and may increase when nervous tone or general strength is lowered. Occasionally tinnitus is slight or absent at certain periods of the day, or absent on one day and loud on another, without any obvious cause for the variation. It may be at first paroxysmal, afterwards continuous. A sudden increase is occasionally met with, and in some cases attacks of labyrinthine vertigo are heralded by increasing intensity of sound, compared even to the whistle of a swiftly passing train.

The effect of external noises may be to increase or lessen the subjective sound. Either condition may be met with in labyrinthine disease. In one such case the tinnitus always ceased with stillness and was excited by sounds; the singing in church (the patient was a clergyman) caused distressing noises. But it is more common for considerable sounds to make slight tinnitus inaudible which is always perceived when there is no noise, and hence many patients suffer much at night. Sounds may not only prevent the tinnitus being noticed; they may actually arrest it. In one patient in whom the tinnitus probably had its source in the auditory nucleus, it was at once stopped if she sounded even a soft musical note with her own voice. The tinnitus came on every evening, and lasted twelve hours; it was a continuous loud rushing noise, referred to the head between the ear and occiput, with no trace of deafness, but with other indications of central disturbance, *e. g.* attacks of numbness in the night in one thigh. In rare instances, loud noises increase the sound and slight ones lessen it, as in a case of bilateral tinnitus with impaired nerve-hearing on one side only.

The state of hearing presents four conditions in these cases. (1) As we have seen, nerve-deafness is common on one or both sides. It may be one-sided when sounds are referred to both ears; when this deafness exists, the morbid process that irritates the auditory nerve also impairs its function, or hinders the passage of vibrations through the ear. In some cases of progressive disease, the deafness gradually increases, while the noises become less, and they may cease altogether when the patient is entirely deaf. In other cases, the noises continue in spite of absolute loss of hearing. (2) Hearing may be perfectly normal to every test. (3) The tinnitus seems to render the patient deaf while it lasts, as we have just seen. This is chiefly met with when the sounds are paroxysmal in existence or intensity. The patient may have "to strain every nerve to hear," and in rare cases there is distinct interference with the action of the auditory centres. In one case, the paroxysms coincided with darting pain through the base of the skull, and distinct word-deafness; words were heard with difficulty, and their meaning could not be made out. There was no evidence of ear disease. (4) There may be increased sensitiveness, hyperacusis, either during attacks of tinnitus or persistent.

A sound produced in an intra-cranial aneurism (of the internal carotid or vertebral) may be heard by another person on listening through a stethoscope applied to the skull. In extremely rare cases, sounds, apparently of intra-aural origin, have also been audible on auscultation, as in the case of a boy, eight years of age (observed by Greene*), in whom an aneurism was most unlikely. In another case, a pulsating sound, resulting from some effect of a blow two weeks before, could be heard eight inches from the patient.†

The sound of muscular contraction within the ear has a peculiar vibratory character, and is probably usually produced by the stapedius muscle, possibly sometimes by the tensor tympani. The former is supplied by the facial nerve, and its central connections are related to those for the orbicularis palpebrarum, so that, in many persons, a strong contraction of the orbicularis is accompanied by this peculiar sound in the ear. The same sound may accompany facial spasm (see p. 254). The function of the stapedius, like that of the orbicularis, is to guard the sense-organ,—to prevent the base of the stapes being driven too far into the foramen by an excessive movement of the membrana tympani. The tensor tympani may have been involved in a case in which a "fluttering" in the ear, for five or ten minutes at a time, accompanied attacks of pain in a case of senile degenerative neuralgia. Clicking sounds are probably due to the action of the muscles connected with the Eustachian tube. These also are sometimes audible by another person.‡ In one case clonic spasm of the levator palati gave rise to such a sound, repeated 120 times a minute, and audible twenty feet from the patient. It ceased only during sleep.* As already stated, tinnitus is often associated with neuralgia, headache, and various manifestations of the "nervous" temperament. The last are often induced by the affection, which, when it interferes with sleep, may have an intensely depressing influence, and it has been known more than once to induce a hopeless sufferer to end his life.

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* Greene, 'Trans. American Otological Society,' 1878.

† Poosten, 'Monatsbl. f. Ohrenheilk.,' 1878, No. 4.

‡ Backer, 'Zeitschr. f. Ohrenheilk.,' xiv, 1885, 237.

DIAGNOSIS.—The characters of the tinnitus thus afford little help in diagnosis, except that, in the rare cases in which the sound is elaborate it is almost certainly of central origin. An aneurism is probable if the sound is audible on auscultation, and certain if this coincides with the symptoms of a basal tumour. But in all cases the chief indication of the cause of tinnitus is afforded by associated symptoms. The most important of these is deafness, which is present in the majority of cases. The cause of the deafness is, as a rule, the cause of the tinnitus. The position and nature of the disease interfering with hearing must, therefore, be ascertained on the principles already described. If must be remembered that disease of the meatus or middle ear can only cause tinnitus by its influence on the labyrinth, either by increasing the labyrinthine pressure or by the extension of the morbid process. If there is evidence of disease, not of the middle ear but of the labyrinth or nerve-trunk, we can only distinguish between these by associated symptoms; if they are absent we must be guided by the fact that disease of the labyrinth is far more frequent, and therefore in any given case more probable, than disease of the nerve, and that this is especially true if deafness co-exists. When tinnitus is due to organic disease of the centres, other definite symptoms are rarely absent, and indicate its seat and nature.

But the questions that most often present themselves are—(1) whether in cases of labyrinthine disease the auditory centres take a share in producing the symptom; (2) when there is no deafness, whether the symptom is wholly central, or (3) is due to irritation of the nerve-endings that does not lessen their function. The last is suggested only when deafness has existed at a previous time, the first when elaborate sounds concur with evidence of labyrinthine deafness. The recognition of primary central tinnitus presents the greatest difficulties. It is only justified when there is no impairment of hearing, when the sounds are elaborate in character, or when simple sounds, referred to the head, occur in a patient who suffers from other central nerve disturbances. Instances of this have been already mentioned (as the case of neuralgic tinnitus, p. 273); it was probable in a young man who suffered from headache, head-pressure, and varied dysæsthesia, in whom the sound was a continuous "hissing" and occasional "humming" or "whistling," and always referred to the head.

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* Williams, 'Zeitschr. f. Ohrenheilk.,' xiii, 1884, p. 99.

Prognosis.—With the exception of the cases in which the noise is due to removable ear disease or to easily remedied constitutional disturbance, the prognosis is very uncertain. In many cases the noises persist in spite of all treatment, but considerable relief is sometimes obtained, and the symptom is removed not unfrequently if the conditions under which it occurs are carefully studied and treated.

Treatment.—The first element is the treatment of any discoverable morbid process on which tinnitus may depend, directly or remotely. This causal treatment comprehends, first, the removal, as far as possible, of any ear disease by which the symptoms may have been produced; and, secondly, the treatment of any general condition with which it may be connected, directly or indirectly,—anæmia, defective nerve-power, gout, syphilis. Tinnitus in gouty persons is often lessened by alkalies and free purgation. In all cases the general health should be carefully attended to, and all influences that increase the noise should be avoided. Those who suffer in consequence of habitual exposure to sounds should obtain rest in a quiet place, and protect themselves by obstructing the meatus. If such causes are not discoverable, or such treatment fails, the symptom itself must be treated, and our power of influencing it is unfortunately very small.

The noise is sometimes diminished by sedatives that lessen nervous activity and over-activity. Of these bromide has more influence than any other drug. In many cases its effect is very marked, although less than on the giddiness which often accompanies the noise. It should be given in scruple doses two or three times a day. Hydrobromic acid has been recommended, but seems to have no advantage over bromide, into which it must be converted as soon as it enters the alkaline blood, while its acidity interferes with its administration in adequate doses.[*] The effect of bromide is sometimes increased by the addition, to each dose, of tincture of belladonna (♏x) or tincture of Indian hemp (♏iij—v). Morphia, by hypodermic injection, lessens the tinnitus for a time, but is only suitable as an occasional palliative when violent paroxysms occur. No other sedative has an appreciable effect on the noises.

Counter-irritation is unquestionably useful. A blister behind the ear often causes the sound to be less loud for a week or ten days after the application, and repeated blisters sometimes produce a permanent diminution in the intensity of tinnitus, although they rarely cause it to cease. The effect is manifest in cases of long duration as well as in those of recent origin, and cannot therefore be ascribed merely to an influence on inflammatory processes.

Drugs that are known to interfere with the functions of the internal ear have been given in the endeavour to alter the morbid action, and in the hope that the disturbance produced may be antagonistic to that of the disease. The only evidence of such an influence is the production of deafness, tinnitus, and vertigo. These are symptoms common to morbid processes of varied character and seat. Politzer noted long ago that subjective noise was often temporarily reduced, together with hearing power, by quinine. Charcot has given quinine in sufficient doses to produce cinchonism, and found that in some cases, when the toxic effect had passed away, the noises were less. I have tried this and also salicylate of soda (15 grains three times a day) in the same way, and have found the influence of the latter more distinct than that of quinine. Of course it is only in chronic and stationary cases that this treatment is admissible, since these drugs cause actual hyperæmia of the labyrinth, and are capable of increasing acute disease.* Lucae has advocated the treatment of tinnitus (especially when the sound has a musical character) by exposing the patient daily for a short or long time, according to circumstances, to a sound of the opposite character to that which he hears,—to a high tone if the subjective tone is low, and *vice versâ*. He employs a tuning-fork, acting on a resonator placed in the meatus.†

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[*] The dose of the solution of hydrobromic acid usually prescribed is 20—60 minims, and this is equal only to 3—8 grains of bromide of potassium.

GLOSSO-PHARYNGEAL NERVE.

The origin of the glosso-pharyngeal nerve is described at p. 45. It leaves the surface of the medulla near the highest fibres of the pneumogastric, and is ultimately distributed to the back part of the tongue, the soft palate, tonsils, upper part of the pharynx (mucous membrane and muscles), to the Eustachian tube, and to the tympanic cavity. The connections of the nerve are important. The tympanic nerve of Jacobson (arising from the enlargement on the glosso-pharyngeal termed the "petrosal ganglion") forms, with the sympathetic, the tympanic plexus in the wall of the middle ear, and gives two branches, one to the large superficial petrosal nerve (from Meckel's ganglion to the facial nerve), and the other (the small petrosal nerve) to the otic ganglion. Thus the glosso-pharyngeal nerve is connected, certainly with the otic, and perhaps also with the spheno-palatine ganglion, of the fifth nerve. Connections with the facial nerve are effected by a branch from the small petrosal nerve to the gangliform enlargement of the facial, sometimes also by a filament to the trunk of the nerve from the digastric branch of the facial, possibly by the connection of the nerve of Jacobson with the large superficial petrosal. It is connected with the pneumogastric at the petrous ganglion, and in the pharyngeal plexus.

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* It has been objected that this treatment is homœopathic. It is not more homœopathic than the treatment of psoriasis by irritants, or the administration of alcohol to reduce the rapid pulse of fever, or the application of an alum lotion in conjunctivitis, or the treatment of the constipation of colic by opium. Besides, to make the production of like symptoms a systematic ground of rejection of treatment, is only less irrational than to make it a systematic ground for the adoption of treatment. To adopt the former system would be to verify the epithet of "allopath," at present an untrue name, invented to conceal, by a factitious contrast, a greater absurdity.

† The method has been praised also by Barr, 'Brit. Med. Journ.,' 1887, ii, 454.

Our knowledge of the functions of the glosso-pharyngeal nerve, and of the symptoms of its paralysis, is less definite than with regard to any other cranial nerve. This is due to the circumstances that the experimental study of its functions in animals is extremely difficult, and that in man it is scarcely ever diseased alone. Hence its functions have to be inferred from its anatomical distribution, and the connection with other nerves lessens considerably the value of conclusions thus reached, because the functions suggested by the termination of its fibres may be in part due to its connections, and may not represent the functions of its root.

The muscular fibres of the upper part of the pharynx are supplied by the pharyngeal plexus, and opinion is divided as to whether the motor fibres come from the glosso-pharyngeal or from the pneumogastric. The glosso-pharyngeal nucleus, however, contains large nerve-cells, motor in aspect, a fact which suggests that it may furnish the fibres for these muscles. Most anatomists think that the fibres to the stylo-pharyngeus come from the facial, by the twig from the digastric branch of the latter. Whether the glosso-pharyngeal innervates any palatine muscle is not known. It is probable that it supplies sensory fibres to the upper part of the pharynx, and perhaps also to the tympanic cavity. It is not probable that it is the sensory nerve for the front of the soft palate, palatine arches, or back of the tongue, since these are rendered anæsthetic by disease of the root of the fifth nerve. But it is generally believed that nausea is produced through the agency of this nerve. It is commonly regarded as the nerve of taste for the back of the tongue, palate, and the fauces. Some even believe that it subserves taste in the front of the tongue, although the unquestionable relation of this to the chorda tympani renders it necessary to assume that this nerve derives its taste-fibres from the glosso-pharyngeal by the connection between the large and small petrosal nerves (see p. 227). The problem of the nerves of taste has been already considered. The fibres of the glosso-pharyngeal have been traced to the circumvallate papillæ, the nerve-structures in which, believed to subserve taste, undergo degenerative changes after division of the nerve-trunk. But there is no instance on record of loss of taste at the back of the tongue from disease of the roots of the glosso-pharyngeal nerve, while there is evidence of its persistence in spite of such disease, and also that disease of the root of the fifth nerve causes loss of taste on the back as well as the front of the tongue, and on the palate and palatine arch. Hence it is impossible to resist the conclusion that if some of the terminal fibres of the glosso-pharyngeal nerve subserve taste, such fibres come ultimately from the fifth nerve. Their path must then be from the otic ganglion, through the tympanic plexus, to the petrous ganglion of the glosso-pharyngeal—a circuitous path, but scarcely more so than that which the taste-fibres of the chorda tympani undoubtedly take. This view is supported by the fact that taste on the back of the tongue may be lost in disease of the middle ear—a fact explicable on no other hypothesis.

The nerve may be diseased by any of the intra-cranial processes that damage the nerve-roots—meningitis, tumours, &c., and its motor fibres participate in the central degenerations, &c., that produce labio-glossal paralysis. It is probable that the pharyngeal symptoms of chronic and acute bulbar paralysis (labio-glosso-pharyngeal paralysis) are due largely to interference with the functions of this nerve. Of the symptoms of its isolated paralysis, nothing certain is known, but it is probable that the upper part of the pharynx is rendered insensitive and weak, so that deglutition is difficult, but both nerves may have to be damaged for the production of these symptoms.

PNEUMOGASTRIC AND ACCESSORY NERVES.

Of all the cranial nerves, the pneumogastric has the most extensive distribution, supplying the pharynx, larynx, lungs, heart, œsophagus, and stomach, and even, in part, the intestines and the spleen. In some of the so-called functional diseases of the organs which it supplies, its action is conspicuously deranged. The symptoms of its disease are thus very extensive, and it will be well first to describe them generally, and afterwards to consider in detail those that merit separate description.

Some of the functions subserved by the trunk of the pneumogastric depend on the fibres which it derives from the spinal accessory; but it is convenient to consider disease of these fibres in connection with that of the root of the pneumogastric, and to describe separately the derangement of the spinal fibres of the accessory nerve which supply the muscles of the neck.

The pneumogastric, it will be remembered, arises from the side of the medulla, between the glosso-pharyngeal above, and the spinal accessory below and to the outer side of the hypoglossal. Its origin is described at p. 45). The trunk of the nerve, after receiving fibres from the spinal accessory, and giving off some small branches (of which the most important is one to the external ear), passes down the neck, behind and in the same sheath with the carotid artery, enters the thorax, on the right side over the subclavian artery, and, on the left, between the subclavian and the carotid; passes through the thorax beside the œsphagus; and ends in branches to the stomach, spleen, and intestines. The most important branches are the pharyngeal, which, with the glosso-pharyngeal, forms the plexus of the same name; the superior laryngeal; the recurrent laryngeal, which passes back, the left around the arch of the aorta, the right around the subclavian artery; branches to the œsophagus; pulmonary branches which, by means of the pulmonary plexus, supply the lung; and branches which form the cardiac plexus for the heart.

The vagus nerve, besides containing motor fibres for the pharynx and larynx, is the chief afferent nerve for the respiratory centre. It contains accelerating and inhibitory fibres for this centre, but the former preponderate, so that experimental division of the nerve in an animal renders the respirations less frequent but deeper, while stimulation of the divided (central) end quickens the respiration, and the acceleration may even proceed to tetanic arrest. The inhibitory fibres are contained chiefly in the superior laryngeal nerve, and their stimulation arrests the respiration, the muscles being relaxed. It is the inhibitory nerve of the heart; slight stimulation increases the diastolic periods, and stronger stimulation arrests the action of the heart. On division of the nerve the cardiac contractions are quickened. It has been said to contain trophic fibres for the heart and lungs, but this is not certain. The pneumogastric is an afferent nerve for the vaso-motor centre, the action of which is lowered by its stimulation, so that the arteries throughout the body are relaxed. It is the motor and sensory nerve for the œsophagus, the sensory nerve for the stomach, and partly also the motor nerve for the stomach and intestines.

Derangement of the Pneumogastric generally.

Causes.—The deep position of the pneumogastric and its branches preserves it from some forms of damage, although its extensive course renders it liable to suffer from many causes. The nucleus in the medulla may be affected by local softening, hæmorrhage, or slow degeneration; but in all these cases other adjacent nuclei suffer also. The nerve, at its origin from the medulla, may be compressed by thickening of the meninges, growths from the meninges or bones, or aneurism of the vertebral artery. Such local disease is often due to syphilis, and the process commonly involves other adjacent nerves at the same time. The trunk of the nerve is sometimes, but rarely, implicated in punctured or gunshot wounds; incised and lacerated wounds in its position are usually immediately fatal from injury to the large blood-vessels to which it is contiguous. In surgical operations the trunk and branches of the nerve are occasionally damaged; the trunk has been tied in ligature of the carotid, and divided in the removal of deep-seated tumours. In such operations in the lower part of the neck it is often also difficult to avoid injury to the recurrent laryngeal. In excision of an enlarged thyroid, both recurrent laryngeals have been repeatedly divided, from the time of Galen down to the present day. Sarcomatous and other tumours, and enlarged glands, may compress or involve the nerve in almost any part of its course; and interference with its function especially occurs from such disease in regions limited by rigid structures, as in the upper part of the neck near the skull, and in the upper part of the thorax. Aneurisms may compress the nerve or its branches; the recurrent laryngeals suffer from this disease with especial frequency. Other causes of interference with the recurrent laryngeals are described in the section on paralysis of the larynx. The vagus is, in rare cases, the seat of neuromata. Neuritis of the trunk of the nerve, due to cold, is supposed to be an occasional cause of symptoms; such cases are extremely rare, but in some acute forms of multiple neuritis this nerve has certainly been involved, since symptoms of its derangement are occasionally produced by toxic influences, which may act on its nucleus, but more probably produce neuritis. The vagus may thus suffer in diphtheritic paralysis, in polyneuritis due to cold, septicæmia, and less commonly to alcohol.

SYMPTOMS due to paralysis of the vagus are more frequently met with than those which result from its irritation. Occasionally both are combined. Laryngeal spasm and vomiting are the irritative symptoms most commonly met with, but occasionally cardiac inhibition occurs. Czermak, was able at will to arrest his heart for a few beats by pressing a small tumour of the neck against his pneumogastric. Concato had a patient in whom a similar inhibition could be caused by pressure on the right nerve. The increased frequency of pulse which corresponds to paralysis of the vagus has been several times noted, and has occasionally been associated with diminished frequency of respiration, although the laryngeal paralysis, also resulting, has usually obscured the effect on the respiratory movements. Roux tied the trunk of the vagus with the left carotid; instantly respiration was arrested; the pulse was also retarded; although the ligature was immediately relaxed, the patient died in half an hour. Robert also tied the nerve with the carotid; the patient, who was conscious, immediately called out, "I am suffocated!" and his voice became hoarse; he recovered, but the hoarseness continued for six months. An instructive example of interference with the functions of the vagus has been recorded by Guttmann. A lad, after diphtheria, presented paralysis of the palate and of one sterno-mastoid. His respirations quickly became reduced to twelve per minute, and were very laboured, while his pulse rose to 120, and he died in a few hours. In many other cases a similar change in the pulse and respiration has been noted, and even a pulse-rate of 160—200. In the face of these observations, and of experiments on animals, it is not easy to understand a fact observed by Billroth, who excised half an inch of one pneumogastric, which was implicated in a tumour, without any resulting symptoms, unless the nerve was previously degenerated.

The important central relations of the vagus, above alluded to, cause derangement of its function to form part of many so-called functional disorders of the central nervous system. Its nucleus forms part of, or is connected with, the respiratory centre, which is conspicuously disturbed in hydrophobia and some other diseases. The phenomena of "Cheyne-Stokes breathing," or "respiration of ascending and descending rhythm," are probably the result of lowered action of the respiratory or pneumogastric centre (see p. 128). The central connections of the vagus, in the cerebral hemispheres, extend to, or are connected with, those parts which are concerned in emotion, and it is apparently through the agency of this nerve that the heart's action is affected in excitement and fear. In many epileptic fits, the central representation of the nerve is the part through which consciousness is first affected, and hence the so-called "epigastric aura."

A similar disturbance seems to be the cause of the globus hystericus and of the laryngeal spasm which are conspicuous in some epileptic and hysteroid seizures. The nerve is closely connected with the centre or nerves for equilibration, so that severe vertigo, on whatever dependent, is often followed by vomiting. The nucleus is contiguous to the internal auditory nucleus, and part of the auditory nerve, that which comes from the semicircular canals, is known to be concerned in the process of regulating the maintenance of equilibrium. In the vertigo that results from disease of this nerve or of the canals (labyrinthine or auditory vertigo) vomiting is very common, and the nausea and retching of sea-sickness are probably due to the deranged action of the semicircular canals in consequence of the motion of the endolymph, which acts on the nerves and deranges the pneumogastric centre. It is possible that the connection of the vagus with the equilibrial nerves is by means of the cerebellum, disease of which so constantly causes vomiting, although this connection has not yet been definitely traced. Conversely, gastric disturbance of the vagus may be accompanied by vertigo, especially when combined with pre existing imperfect action of the fibres from the semicircular canals.

PHARYNGEAL BRANCHES.

Branches of the pneumogastric form, with the glosso-pharyngeal, the "pharyngeal plexus." From this plexus the muscles and mucous membrane of the pharynx are supplied. The special distribution of the branches of each constituent nerve is not known.

The most common cause of paralysis of the pharynx is disease of the origin of the nerve in the medulla; such disease commonly also involves adjacent nuclei. Paralysis may, however, result from meningeal disease outside the medulla, and from disease of the bones of the base of the skull, but it is scarcely ever due to disease outside the skull. It occasionally forms part of diphtheritic paralysis.

PARALYSIS of the pharynx is manifested by difficulty of swallowing; food, entering the pharynx from the mouth, lodges there instead of descending to the œsophagus. Small particles or liquid may enter the larynx, and cause spasm and even actual choking. Pulpy food can be swallowed better than solids or liquids. If the paralysis is limited to the superior constrictor, liquids may, it is said, be forced into the nose by the contraction of the middle constrictor, but it is doubtful whether this occurs unless the soft palate is also paralysed. The affection of the nerves on one side causes only slight difficulty in deglutition, no doubt on account of the circular arrangement of the muscular fibres.

Paralysis of the pharynx can be confounded with other conditions only in consequence of imperfect observation. I have known the difficulty in swallowing to be attributed to malignant disease, but such a mistake ought not to occur. If any doubt exists as to whether dysphagia is due to paralysis or organic obstruction, the passage of a sound will at once exclude the latter. From pharyngeal spasm, paralysis is distinguished by its continuous character.

SPASM of the pharynx is always part of "functional" disturbance, and does not result from organic disease, in the usual sense of the word. It is often due to hysteria, and probably occurs in the more severe form of hysterical "globus." It is usually associated with other hysterical symptoms, but sometimes occurs alone. As an instance may be mentioned the case of a gifted but highly nervous man, who for many years could only swallow food when alone; in the presence of others, spasm of the pharynx always prevented deglutition. Similar spasm occurs during the paroxysms of hydrophobia.

LARYNGEAL BRANCHES.

PARALYSIS OF THE LARYNX —Paralysis of the larynx is a large subject, of which only the more important outlines can be given here. The organ is innervated by two branches of the vagus. (1) The superior laryngeal, which arises high up in the neck. It is the sensory nerve of the larynx above the vocal cords, and supplies the crico-thyroid muscle and the deflectors of the epiglottis. (2) The inferior or recurrent laryngeal, which arises in the upper part of the thorax, and passes up to the larynx, between the trachea and œsophagus. It gives sensibility to the larynx below the vocal cords and to the whole trachea; and it supplies all the muscles of the larynx except the crico-thyroid and epiglottidean muscles. All the motor fibres for the larynx come from the spinal accessory; only the sensory fibres pass to the medulla by the roots of the pneumogastric itself.

In order to understand the symptoms of laryngeal paralysis it is necessary to have a clear conception of the anatomy of the larynx, and of the action of its muscles. It may be well, therefore, first to enumerate, in brief outline, the facts which are of the most salient importance.

The glottis is opened or closed by the movement of the posterior extremities of the cords only (the anterior remaining always fixed), and this movement is effected chiefly by the motion of the arytænoid cartilages. These cartilages are attached to the cricoid cartilage by an articulation that permits free movement. Each has the form of an irregular pyramid, prolonged at the base into two processes, an anterior or vocal process, from which the vocal cord extends to the thyroid cartilage, and an external or muscular process, to which the muscles are chiefly attached. When the latter, which is at right angles to the vocal process, is moved back, the vocal process moves outwards, away from its fellow; the cord is abducted, and the glottis opened. When the muscular process is moved forwards, the vocal process is moved inwards towards its fellow; the cord is adducted and the glottis closed. But these movements of the cords are further aided by the movement of the arytænoid cartilages away from or towards each other.

The most important muscles of the larynx and the effects of their palsy may be briefly described.

Crico-thyroid.—The fibres, outside the thyroid cartilage, pass downwards and forwards to the cricoid, which they draw back and slightly tilt, lowering the posterior part of the cartilage with the attached arytænoids, and thus they elongate and make tense the vocal cords. Isolated paralysis is singularly rare, and there is much uncertainty about its exact symptoms. It is said to impair the production of high notes, but if so, it is probably because, without the influence of the crico-thyroid, the internal thyro-arytænoideus cannot act with effect. It is probable that paralysis influences the height of the cords during the production of high notes, but this effect can only be recognised when the paralysis is one-sided; the arytænoid cartilage and vocal cord are higher on the paralysed side than on the other, on account of the obliquity of the cricoid cartilage, and the posterior part of the glottis is displaced towards the paralysed side (Riegel).

Thyro-arytænoid.—The fibres pass backwards, from the posterior surface of the front of the thyroid cartilage, close to and parallel with the vocal cord. The inner fibres are connected with the cord, and seem to influence the distribution of tension. The outer fibres pass to the muscular process of the arytænoid, and, if this cartilage is free to rotate, they draw the outer process forwards, and the vocal process inwards, thus adducting the cord. The inner fibres, and also the outer, if rotation is prevented, shorten the cord. Paralysis of the inner fibres renders the cord atonic with a concave edge. Paralysis of the outer fibres doubtless lessens the power of adduction, although in a degree that is difficult to recognise.

Lateral crico-arytænoid.—The fibres pass from the side of the cricoid backwards and upwards to the outer process of the arytænoid cartilage, and, drawing this forwards, move the vocal process inwards and adduct the cord. It is the chief adductor. Isolated paralysis is very rare, but would certainly lessen considerably the power of adduction ; it is probable that the cord could still be brought to the middle line by the thyro-arytænoid, unless there was secondary contracture of the abductor.

Arytænoid.—This, passing between the arytænoid cartilages behind, draws them together and thus adducts. In isolated palsy the vocal cords are brought together, except behind, between the arytænoid cartilages, where a small triangular space remains.

Posterior crico-arytænoid.—The fibres of the "posticus" muscle pass from the posterior surface of the cricoid cartilage outwards, upwards, and ultimately forwards, over the edge of the cricoid cartilage to the muscular process of the arytænoid cartilage. They draw this back and the vocal process outwards, and thus abduct the cords and open the glottis. It is the only special abductor, and is thus a muscle of great importance. When it is paralysed alone, abduction is impossible, and the vocal cord is in the middle line, adducted in consequence of the unopposed action of the adductors.

Thus the vocal cord is abducted chiefly by one muscle, but the widening is increased by the separation of the arytænoid cartilages themselves, effected probably by the simultaneous action of the posterior fibres of the lateral and outer fibres of the posterior crico-arytænoids. These fibres draw the arytænoid cartilage downwards and outwards on its convex articular surface. The cord is adducted, and the glottis closed, by several muscles—by the lateral crico-arytænoid and the outer part of the thyro-arytænoid, which rotate the cartilage, and by the arytænoids, which bring the cartilages together. The vocal cords are lengthened and made tense by the crico-thyroid, shortened by the thyro-arytænoids, and made either lax or tense in parts, according as the inner fibres of this muscle are inert or active.

The muscles must, however, act in very complex combinations. The different fibres of each muscle have not all the same direction, and cannot have the same action. The difference may even be such that some fibres of one muscle may have an effect opposed to that of the rest, if they act alone or with fibres of another muscle. This is illustrated by the fact, already mentioned, that the posterior fibres of the lateral crico-arytænoideus, acting with the outer fibres of the posterior crico-arytænoideus, may aid in abduction by drawing the arytænoid cartilage downwards. On the other hand, if the other glottis-closers are acting powerfully, the highest (horizontal) fibres of the posterior crico-arytænoideus, the chief glottis-opener, may even aid closure by helping to approximate the arytænoid cartilages. If the action may be thus complex in simple opening and closing the glottis, how much more complex must it be in the delicate and varied actions by which is produced the infinite variety of vocal sounds!

CAUSES.—Paralyses of the larynx fall into several widely different categories, whether they are considered in reference to their symptoms or to their causes. The chief causes are—

(1) Organic disease of the centres, or of the nerves outside the larynx. According to the seat and character of the disease the palsy may involve one or another group of muscles, and may be unilateral or bilateral.

(*a*) A rare cause is disease of the cortical centre for the larynx in the lowest part of the ascending frontal convolution (Semon and Horsley). Such disease, to be effective, must be bilateral, because the loss of function of one centre is compensated by the other; "pseudo-bulbar paralysis" results from disease on both sides, with the symptoms described in the account of bulbar paralysis. Bilateral disease of the conducting tracts in the hemispheres is equally effective.

(*b*) The most frequent central cause is chronic nuclear degeneration, involving the cells of the highest part of the spinal accessory. In rare cases the lesion is acute nuclear softening. In either case the symptoms are usually associated with paralysis of the tongue and lips, as labio-glosso-laryngeal paralysis," chronic or acute; it will be remembered that the highest part of the spinal accessory and the hypoglossal nuclei are contiguous, the former behind, the latter in front of the central canal of the cord. Palsy from chronic degeneration is often associated with wasting in other muscles. Degenerative paralysis of the larynx occurs sometimes in disseminated sclerosis, in general paralysis of the insane, and especially in locomotor ataxy. After diphtheria, also, the larynx may be paralysed, in consequence of central changes or neuritis. The palsy in most cases is bilateral, and usually abductor; it is unilateral only in rare instances. Some other toxic blood-states have a similar effect, and it is probably by this mechanism that such paralysis occurs in "acute ascending paralysis."

(*c*) Damage to the roots of the nerves is also an occasional cause, commonly of syphilitic origin, sometimes the result of an aneurism or other simple tumour. Paralysis from this cause is usually one-sided, and often affects half the tongue and palate as well as the vocal cord.

(*d*) The long course of the nerve-trunks exposes them to damage from many morbid processes and injuries, which have been enumerated in the section on general causation, and to these must be added the causes of multiple neuritis, in which the laryngeal nerves are occasionally involved, early or late. From local causes the superior laryngeal nerve is in general (on account of its course) less liable to suffer than the recurrent. Hence disease of the latter is the most frequent cause of such laryngeal palsy. Aneurism of the vessels, round which the nerves turn, frequently compresses them; and the left suffers from this cause more frequently than the right, on account of its course round the aorta. Other causes of pressure are growths and enlarged glands in the thorax, cancer of the œsophsgus, and enlargement of the thyroid. Paralysis has been met with in some cases of chronic lung disease, and has probably been produced through the agency of enlargement of the glands; the paralysis has been unilateral or bilateral. In all these cases, except disease of the superior laryngeal, many muscles are of necessity paralysed, although the resulting symptoms are not always the same.

(2) The laryngeal muscles are often weakened by processes that, involving no organic change in nerve or centre, and passing away completely in many instances, are termed "functional." The morbid process may be central or local in its conspicuous relations, although we cannot affirm that these always indicate correctly the seat of the morbid process. The chief central causes are hysteria and the nervous weakness that attends anæmia and follows prostrating maladies. The local causes are congestion and inflammation of the mucous membrane, and over-use of the larynx, especially in public speaking. We cannot altogether separate the two classes, since local and central disturbance may coincide in cases that appear to be of local origin; hysterical aphonia, for instance, is frequently excited by a transient laryngeal catarrh. Moreover some cases, now regarded as functional, are probably due to slight forms of polyneuritis, some toxic causes which have more tendency to affect this than other nerves. Paralysis of the larynx has also been thought to be occasionally reflex in origin. Thus abduction-palsy, necessitating tracheotomy, has developed without traceable cause during pregnancy, and has passed away after delivery.*

(3) Paralysis of a single muscle on one side results only from local disease affecting the minute branches within the larynx. Laryngeal growths, cellulitis, and ulceration of the cartilages are occasional causes; but palsy of this nature is not common. Cold has been supposed to excite a rheumatic neuritis of these branches, but when this cause is effective other nerves usually suffer, and the case comes into the category of peripheral polyneuritis.

(4) Lastly, cases of laryngeal palsy are sometimes met with for which no adequate or probable cause can be discovered. Such cases will doubtless lessen in number as our knowledge increases.

Symptoms.—The larynx is the organ of voice, and the gate of the air-passages. By the aid of the laryngoscope its action in these two functions can be directly observed. The evidence of paralysis is correspondingly threefold. (1) Voice may be changed or lost; (2) the entrance of air in respiration may be impeded, or the closure of the glottis in cough may be impaired; (3) the defective movement can be, in part at least, directly observed. The phonic and respiratory functions are subserved by the same muscles and the same nerves, but by centres that no doubt differ in anatomical connection if not in position.

The vocal cords assume, after death, a position of slight abduction from the middle line,—a little nearer together than they are during ordinary breathing. This position must, therefore, be regarded as that of muscular relaxation,—of the rest that, during life, they may approximate, but never actually attain. During phonation the cords are brought together and made tense; the degree of approximation and tension varies according to the note produced. In breathing the cords are separated during inspiration, the extent of abduction being proportioned to the force of the inspiration; during expiration they are brought a little nearer together than the cadaveric position.

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* Aysaguer, 'L'Union méd.,' March 31st, 1885.

If there is complete paralysis of all the muscles, or (what is the common condition) of all except the crico-thyroids, the vocal cords assume the cadaveric position, from which they cannot be moved (Fig. 109). They are not approximated on an attempt to phonate, nor do they recede on deep inspiration. Hence vocal sounds cannot be produced. In deep inspiration the current of air may bring the cords a little nearer together than normal, and may cause slight stridor. Instead of the natural explosive cough there is only a sudden rush of air through the glottis. If only one cord is paralysed, this cord alone is motionless in the "cadaveric position" (Fig. 110), the other moves normally. Some phonation may still be possible, because the unaffected cord may be over-adducted beyond the middle line, but the voice is low-pitched, and often hoarse. The abduction of the healthy cord during inspiration prevents stridor. But, unless the palsy is slight, the glottis cannot be closed with sufficient firmness to effect an explosive cough. Complete paralysis is met with in central disease, in disease of the trunk of the vagus, or in disease of the recurrent laryngeal. The escape of the crico-thyroid in the latter case does not materially modify the condition of the larynx.

FIG. 109.

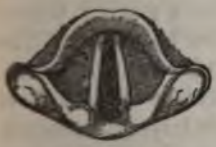

FIG. 110.

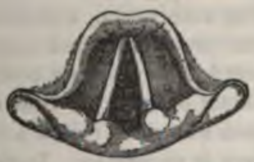

FIG. 111.

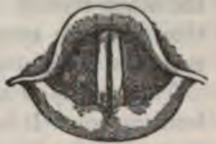

FIG. 109.—Total palsy of both cords. Cadaveric position.
FIG. 110.—Similar palsy of left vocal cord.
FIG. 111.—Bilateral abductor paralysis.

In other cases of paralysis, instead of complete loss of movement with the cords in the cadaveric position, they are nearer together and cannot be abducted, even as far as the cadaveric position (Fig. 111). They can be brought closer together in phonation and cough, and when the effort ceases they recede a little, but the normal wide recession during inspiration does not take place. This indicates paralysis of the abductors, the posterior crico-arytænoids. The slight recession that may occur is due to the elasticity of the attachments of the cords

The position of the cords, and the amount of recession, depend on the duration of the paralysis. The adductors, unopposed, undergo secondary contracture, and with this the glottis becomes permanently narrower, until there may be only, at widest, a narrow chink between the cords. The tensors are still active as well as the adductors, and hence voice is little affected. The cords are indeed always in the position for phonation. The chief symptom is the effect on respiration.* The normal recession of the cords during inspiration does not take place, and they are even brought still nearer together by the pressure of the inrushing air. Hence inspiration is accompanied by a whistling stridor, often very loud; the hindrance to the entrance of air brings into action the extraordinary muscles of respiration, and inspiration occupies a longer time than normal. On account of the shape of the glottis expiration is unimpeded; the current of air even tends to separate the cords. The absence of any affection of the voice often causes an error in diagnosis, and the obstruction is referred to the trachea. But the symptoms differ from this in the absence of expiratory stridor, and the movement of the larynx up and down during breathing is greater than is ever seen in tracheal stenosis. The symptoms are indeed so characteristic that the diagnosis can be made with certainty, even without the aid of the laryngoscope, and they are so striking that, once witnessed, they can scarcely be mistaken, and can never be forgotten. The urgent dyspnœa and loud stridor, as each breath is drawn, accompanied as they may be with lividity of the face, and coldness of the extremities, are alarming. The danger is not apparent only; the slightest catarrhal swelling of the cords suffices to occlude the narrowed glottis, and prompt laryngotomy alone may save the patient from death by suffocation. This account is true, however, only of bilateral palsy. If the paralysis is unilateral, the affected cord is near the middle line and motionless; the other recedes during respiration, sometimes to a greater degree than normal: symptoms are slight, and may even be absent. The recession of the unaffected cord prevents both stridor and dyspnœa.

This paralysis of abduction may be due to central disease or to local causes. It has been known to follow a simple laryngeal catarrh. In some cases its etiology is uncertain. Both posterior muscles have been found destroyed by degeneration when all the other laryngeal muscles were healthy. It is possible that these obscure cases are also of central or toxæmic origin. But a similar paralysis of abduction may be produced by disease of the recurrent laryngeal, although this nerve contains fibres for the adductors as well as the abductors. Instead of the cadaveric position of the cord, and the complete immobility found in many cases of disease of the recurrent, the cord is near the middle line; further adduction is still possible, but there is no abduction, and if both nerves are affected the symptoms above described are present in characteristic degree. Much ingenuity has been expended in attempting to explain why the abductors should suffer chiefly from disease, such as the pressure of an aneurism, which presumably affects all the fibres of the nerve. Some have suggested that the adductors must be supplied also by the superior laryngeal—an hypothesis unsupported by anatomy. It has also been suggested that the fibres for the abductors may be superficial in the nerve, and so suffer first, or have some mysterious special proclivity to disease. Others have remarked that the abductors suffer most, just as the abductors and extensors of a limb suffer more in hemiplegia than the adductors and flexors—a doubtful analogy which affords no explanation.*

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* Hence this palsy was termed by Türck "respiratory paralysis," as distinguished from "phonic paralysis," in which the voice is affected, while the conditions in which both functions are disturbed have been termed "mixed paralysis." This somewhat loose division hardly deserves the wide adoption it has obtained.

The fact that disease of the recurrent laryngeal, affecting all fibres apparently equally, may influence abduction more than adduction, must be taken in connection with another fact, that electrical stimulation of the recurrent nerve causes adduction of the cord. In the former case we are justified in presuming that all the muscles supplied under-act, in the latter case it is certain that all over-act; in each case there is adduction. It is probable that the explanation will be found in the relative bulk and arrangement of the two sets of fibres. The adductors are more numerous than the abductors, are probably more bulky, are certainly more varied in arrangement, and perhaps act at greater mechanical advantage. The direction of the fibres of the posterior crico-arytænoids seems less favorable to their action on the arytænoid cartilages than that of the lateral muscles, and when the cords are adducted the relative disadvantage of the posterior muscles must be increased. Thus an equal reduction of absolute power may cause a far greater impairment of the abductors than of the adductors. Hence it is better to speak of this condition as an impairment or loss of abduction, rather than as paralysis of the abductors. The abduction palsy with narrow glottis probably results from damage to the recurrent nerves, which is incomplete in degree although not necessarily partial in distribution. The complete palsy with cadaveric position of the glottis is the consequence of complete paralysis of the nerve. Thus the latter has been observed to follow the former as the disease progressed (Schech, Rosenbach).

It has been suggested (first by Riegel) that the adduction in disease of the recurrent might be the result of secondary contracture in the unopposed crico-thyroid, which, as a tensor and elongator of the cords, must tend to bring the cord and vocal process into a straight line. The chief difficulty in accepting this explanation is that the adduction is not invariable, as it should be were it the result of a secondary contracture of the unopposed crico-thyroid. Nevertheless secondary contracture may play a part in the phenomena of some cases. If the narrow glottis ever persists after palsy has become complete, it may be from secondary contracture and shortening of the adductors, perpetuated by tissue changes in them.*

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* See Semon, 'Arch. of Laryngol.,' 1881; and Russell, 'Proc. Roy. Soc.,' 1892.

Disease of one recurrent nerve usually affects only the corresponding vocal cord. Paralysis of both cords, when produced by an organic lesion, is generally due to disease of both recurrent nerves or to central changes. But three cases are on record in which both cords were paralysed, although only one recurrent nerve was found to be compressed or otherwise diseased.† In two cases the paralysis was greater on the side of the lesion than on the other. The paralysis of the opposite cord is supposed to be due to an influence on the centre.

Paralysis of abduction also occurs in hysteria, although rarely; it is then always bilateral. The characteristic symptoms are present—inspiratory stridor, often intense, with unimpaired phonation. Most recorded cases have recovered, but the inspiratory dyspnœa may be alarming in its intensity, and it is probable that death has occurred in more than one instance. In a case of morphia habit and hysteria‡ the patient suffered from loud inspiratory stridor, absent during expiration, not interfering with phonation, and due to abductor paralysis. It disappeared entirely while the patient was under the influence of morphia, but reached an alarming intensity when morphia had been withheld for twelve hours, the stridor being audible all over the house. Amaurosis and some mental derangement coincided with the stridor. She recovered rapidly under treatment, which consisted in a modification of the Weir-Mitchell system, the gradual withdrawal of morphia, and the hypodermic injection of strychnine. The abductor paralysis has been mistaken for spasm, on account of the stridor that accompanies it; but the circumstances that expiration is noiseless or nearly so, and speech unaffected, effectually distinguish it from spasm. It is probable that many cases of so-called hysterical spasm of the glottis have been really abductor palsy.

In adductor paralysis the cords are apart and cannot be brought together. The state in which the cords are in the cadaveric position, and cannot be moved, should not be spoken of as adductor paralysis, because, although the adductors are paralysed, all the other muscles are paralysed also. The term should be applied only to the cases in which there is still the power of abduction on deep inspiration, but no power of bringing the cords nearer together than the cadaveric position. Thus defined, adductor paralysis is rarely due to organic disease of the nerves or centres.* It is most common as a partial paralysis; the cords are not brought together in phonation, and the patient is therefore voiceless, but they can be brought together in coughing. Hence this has been termed "phonic paralysis" (Türck). It is common in hysteria, in which it causes the so-called "hysterical aphonia," and it results also from over-use of the voice and from catarrhal laryngitis. It is also said sometimes to result from cold without laryngitis (Bose). In hysterical aphonia the patient is sometimes able to sing, although she can only speak in a whisper (v. Bruns).

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* Wagner attributes the cadaveric position to the crico-thyroid, because it is assumed when the superior laryngeal nerve is divided after the inferior ('Virch. Arch.,' Bd. cxx, p. 437, 1890).

† Bäumler, Johnson, Sommerbrodt. The case of Sommerbrodt was one of traumatic damage.

‡ Seen with Mr. W. L. Winterbotham, of Bridgwater. The diagnosis of the laryngeal condition was afterwards confirmed by Sir Morell Mackenzie.

Another form of partial adductor palsy is due to loss of power in the arytænoideus; the result is defective closure of the posterior part of the glottis and hoarseness or loss of voice. Little is known of the symptoms and causes of paralysis of the tensors of the vocal cords, beyond the fact that palsy of the internal fibres of the thyro-arytænoideus causes the edge of the cord to be concave. The effect on the voice is similar to that of the preceding palsy, and the two are sometimes associated.

For the exact diagnosis of these laryngeal palsies an examination by means of the laryngoscope is essential. But it may be useful to compare the symptoms presented by the chief forms of paralysis, since they often suggest very clearly the nature of the affection. In the following table the symptoms are enumerated in the first column, the condition seen with the laryngoscope in the second, and the form of paralysis in the third. The inability to effect an explosive cough is of great significance as evidence of a palsy of organic origin, in the absence, of course, of local organic disease. Entire loss of voice, as well as of cough, suggests bilateral palsy of grave organic nature, but without loss of cough it indicates unimportant adduction palsy. Loss of cough without loss of voice suggests paralysis of one cord. Loud inspiratory stridor without loss of voice means abduction paralysis.

Symptoms.	Signs.	Lesion.
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low-pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy.
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.

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* Navratil, 'Berlin. klin. Wochenschrift,' 1869, Nos. 36 and 37. The same fact has been recently emphasised by Dr. Semon, 'Arch. of Laryngology,' loc. cit.

Symptoms.	Signs.	Lesion.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line, not moving during inspiration; the other normal.	Unilateral abductor palsy.
No voice; perfect cough; no stridor or dyspnœa.	Cords normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

Anæsthesia of the larynx is rare. It results from disease of the superior laryngeal nerve, or the roots of the vagus; it is occasionally met with in degenerations of the medulla, but is then usually incomplete. It may form part of hemianæsthesia of hysterical or cerebral origin. In the latter cases reflex action is unimpaired; in disease of the nerve or medulla it is lost. General anæsthesia about the entrance to the larynx is said to have been met with in hysterical aphonia.

SPASM OF THE LARYNX

The common form of spasm of the laryngeal muscles is that of the adductors. The muscles that close the glottis are more powerful than those that open it, no doubt because firm closure is necessary for the process of coughing, and in order to fix the thorax during muscular effort. Moreover the reflex mechanism is connected chiefly with the glottis-closers, doubtless to protect the air-passages from foreign bodies, or to aid in the expulsion, by coughing, of any irritant substances that have gained an entrance. Hence any nerve irritation, direct, central, or reflex, causes closure; and it is not surprising that spasm accompanies a large number of laryngeal diseases, varying in its prominence according to the irritative nature of the disease and the excitability of the reflex mechanism. The latter is far more intense in children than in adults, and hence in them the slightest laryngeal catarrh gives rise to spasm. Attacks occur especially at night, when the reflex mechanism, released by sleep from the control of the higher centres, is in its most active state. Spasm may occur from irritation, not only of the superior laryngeal nerve, but also of the vagus below, as when the latter is compressed by a tumour in the upper part of the chest; the afferent impression is due to irritation of the sensory fibres from the trachea. Reflex spasm is always bilateral. Direct spasm from irritation of one recurrent laryngeal usually involves only one vocal cord, but in a few cases spasm, so excited, has been bilateral. This may be explained either by the irritation of some afferent fibres, or (according to Krishaber) by spasm of the arytænoideus, which is a bilateral muscle.

Simple spasm occurs in rickety children, in whom the nervous system is in a condition of excessive reflex excitability. In this form, which is termed "laryngismus stridulus," the vaso-motor and cardiac centres of the medulla are also deranged; the child on some exciting cause, as a start, a peripheral impression, or even without apparent cause, suddenly turns pale, is unable to get its breath for a few seconds, and then, the spasm relaxing, air is drawn through the slowly opening glottis with a crowing noise. Paroxysmal attacks of laryngeal spasm sometimes occur in adults, usually in the night. They are apparently analogous to attacks of asthma, the spasm affecting the larynx instead of the bronchial tubes. The sufferer wakes up with a feeling of suffocation, intense difficulty of breathing, and loud laryngeal stridor, which after a few minutes passes away. During the spasm the distress may be extreme, the patient tears open his clothes, and may seem at the point of death. Such occasional attacks have been known to recur from time to time during many years. They have been known to replace attacks of migraine (Liveing), and are occasionally met with in the subjects of locomotor ataxy, in whom they have been termed "laryngeal crises" (see vol. i).

The paroxysms of laryngismus stridulus probably differ only in degree from the general convulsions that are also common in rickety children. In most epileptic convulsions there is laryngeal spasm, which causes the initial "epileptic cry." During the paroxysms of hydrophobia there is also spasm of the glottis.

Lastly, spasm of the larynx is met with in certain general neuroses, in tetany[*] rarely, in hysteria occasionally. In the latter it may occur in paroxysmal or more continuous form. The paroxysmal form constitutes one variety of hysteroid convulsion. Instances of this are described in the chapter on hysteria. The continuous form is very rare. There is stridor with inspiration and expiration, the voice is feeble, and there may be hysterical rapid breathing. The diagnosis from hysterical abduction-paralysis rests on the fact that the stridor in spasm accompanies inspiration as well as expiration, and the voice is more altered than in abduction-palsy, in which also the loud whistling inspiration contrasts with the almost noiseless expiration. The laryngeal symptoms usually partake of the character of the other disturbances that may be present; the spasm is accompanied by other spasmodic or convulsive symptoms; the paralysis, by loss of power or of sensibility. It is probable that some cases of supposed spasm have been really instances of abduction-paralysis.

A rare condition of functional spasm has been described,[†] in which spasm is excited by attempts to speak. It is, so to speak, the converse of phonic paralysis. In the latter the cords cannot be brought together in speaking; in the functional spasm they are brought together too forcibly. Either the patient cannot speak or speaks at first in an altered voice, which ceases altogether when a greater effort is made, on account of the increased spasm that the effort induces. Apart from attempts to speak there is no laryngeal disturbance. It has been termmed "spastic aphonia" or "phonic laryngeal spasm" by Schnitzler, "spastic dysphonia" by Schech, and "co-ordinated laryngeal spasm" by Nothnagel, because he observed it to accompany other voluntary movements of the larynx besides those of speech, while it was absent in all involuntary and automatic movements. The spasm may be attended with pain in the larynx and even in the upper part of the thorax (Jurasz). The affection has been compared to writers' cramp, but differs from this in its general etiological relations, and in its greater amenability to treatment. A closer analogy to writers' cramp was presented by a case recorded by Gerhardt, in which the patient had actually suffered from writers' cramp, and, at the age of fifty, learned to play the flute. The act of blowing the flute brought on laryngeal spasm and an unintended voice-sound, accompanied by muscular contractions in the arm and angle of the mouth.

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[*] Killian, 'Monatsschr. f. Ohrenkr., &c.,' 1884.

[†] Schnitzler (1875), Schech (1879), Nothnagel (1881), Fritsche, Jurasz (1880).

Various forms of cough occur as isolated symptoms, but scarcely come into the province of this book. One form, however, deserves special mention, the barking cough which occurs in boys about the time of puberty, which is a source of great annoyance to the friends of the patient. It is generally the result of the habit of masturbation, and a knowledge of this enables a case which has gone on for months to be quickly cured.

PULMONARY BRANCHES.

The influence of general disturbance of the pneumogastric on the respiratory movements, and the spasm that results from irritation of the afferent pulmonary nerves, have been already described. The muscular fibres of the bronchi are supplied by this nerve, and their paroxysmal contraction in asthma has been thought to be produced through its agency. It has also been asserted that the plain muscular fibres said to exist in the pulmonary tissue are supplied by the pneumogastric (Gerlach), and their contraction has been assumed to explain a peculiar form of emphysema observed in a case of compression of the pneumogastric (Tuczek); but the compression caused also deep breathing of a costo-superior type, and the emphysema may have been merely the result of the energetic movement consequent on the stimulation of the respiratory centre. The pneumogastric is commonly believed to contain vaso-motor fibres for the vessels of the lungs, but Brown-Séquard and Franck have separately shown that these fibres are contained, not in the vagus, but in the sympathetic. Vascular lesions of the lungs have, however, been observed after section of the vagus. Michaelson noted rapid congestion and hæmorrhage; it is possible that this may have been of reflex origin, produced through the agency of the sympathetic. But in man, acute lesions of the pons sometimes cause rapid vascular changes in the lungs; in one case of hæmorrhage into the pons, fatal in two hours, I found intense congestion with extravasation into the left lung, and hæmorrhages in the left extremity of the stomach. After section of the vagus, animals die from chronic pneumonia, and hence the vagus has been supposed to be a trophic nerve for the lungs. Such changes have also been accounted for by the entrance of food from the pharynx into the bronchi, in consequence of paralysis of the œsophagus and the paralysis of the larynx (Traube, Steiner). All admit that this is one cause of the pulmonary affection, but opinions differ as to the extent of its influence.

It is possible that the curious phenomena of hiccough may be connected with the affections of this nerve, as they certainly are with the respiratory centre. Other paroxysmal attacks of varied and various character are also met with. Thus a man aged thirty-seven, whose family was liable to asthma in the last generation, suffered for one and a half years, at intervals of four days to a week, from peculiar attacks lasting from twelve to twenty-four hours, and characterised by prostration, weakness of the limbs, and pallor of the face with muscular "working" in the chest, followed by a copious flow of saliva, such as precedes vomiting, and then an intense sense of suffocation.

CARDIAC BRANCHES.

The inhibitory effect of irritation, and the acceleration of the heart's action that results from lessened action of the vagus, have been before alluded to. Increased frequency has been several times observed in cases of local disease of the vagus in the thorax, compression by mediastinal tumours, &c. In a case of phthisis, for instance, in which the pulse was unduly frequent (130—148), at first occasionally, and afterwards constantly, Meixner found the left vagus enclosed in a mass of enlarged glands in the upper opening of the thorax. The vagus is also the afferent nerve from the heart; and although we are normally unconscious of the cardiac action, some of the disordered sensations of disease are apparently produced through its agency. In certain anginal attacks the heart's action is, for a time, arrested or retarded, and in a few cases these symptoms have been found associated with organic disease of the cardiac plexus. Thus in a case in which, during paroxysms of intense anginal anguish, the heart's action was arrested for four or six pulsations, Heine found a tumour involving the cardiac plexus. In a case recorded by Blandin, anginal attacks were associated with a small tumour of the vagus. Pseudo-anginal symptoms have been supposed to be associated with neuritis of one vagus nerve.* Further, there are afferent fibres from the heart, inhibiting the action of the vaso-motor centre, and these are probably disturbed in some anginal seizures.

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* See Obolensky, 'Berlin. klin. Wochenschr.,' No. 52, 1889.

The inhibitory fibres also for the heart seem to be sometimes stimulated from the centre, *e. g.* by toxic blood-states having a special influence on this part. But they are occasionally disordered in paroxysms which seem to be related to those of migraine, and like migraine, may be remotely connected with inherited gout, which is a potent cause of functional nerve derangement.*

After disease or injury of the vagus the heart has been found in a state of fatty degeneration, and hence it has been thought that the vagus contains trophic fibres for the cardiac substance.

GASTRIC BRANCHES.

The branches to the œsophagus are rarely diseased except in cases of affection of the nerve-trunk or of the centre. In very rare cases, such disease has caused difficulty in swallowing, simulating stricture. Spasm of the œsophagus is far more frequent. The vagus is the sensory nerve for the stomach. Its fibres are very sensitive to any local irritation, and not rarely the seat of spontaneous neuralgia. Hunger is generally believed to be a pneumogastric sensation, and complete loss of the sensations of hunger and thirst was noted in a case of softening of the root of the vagus from an aneurism of the vertebral artery (Johnson). Appetite, however, is not always lost in animals when the pneumogastrics have been divided (Reid). In some cases of disease of the nerve, excessive appetite has been present. This symptom, for instance, was noted in one case, in conjunction with dyspnœa, noisy breathing, and vomiting of unaltered food; post mortem both pneumogastrics were found atrophied (Swan). In another case of insatiable appetite, small neuromata were found on the nerve. It is possible that the symptom may be partly the result of the defective digestion of food.

The pneumogastric is also in part the motor nerve of the stomach; after its section the contractions of the organ are lessened, although not altogether arrested. Vomiting is probably produced through its agency, by varied reflex and central irritation. In the latter case (as in meningitis) the vomiting is sometimes extremely rapid. I have known paroxysmal vomiting to result from the intermitting pressure of a tumour on the vagus; and Boinet, having exposed the vagus in an operation in the neck, noted that whenever he touched the nerve the patient vomited.

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* A woman, in the course of five years, had six attacks, in which there was a peculiar œdematous erythema in the region supplied by the first division of the fifth nerve on both sides, followed by prolonged retardation of the heart down to 22 and 32 a minute for weeks together, in consequence of prolongation of the systole. During the first few days of each attack the temperature was raised to 102·5°. There was a family history of gout, and the patient had formerly suffered from distinct migraine

The vagus accelerates the contraction of the intestines, but no intestinal symptoms have been observed from its disease.

PROGNOSIS.—The prognosis in every case depends chiefly upon the nature of the disease that interferes with the function of the nerve. It is good only when the disturbance is not due to organic mischief. In the diseases in which the local symptoms form part of a wider functional disturbance, as in hysteria and multiple neuritis, the prognosis is described in the account of those diseases.

DIAGNOSIS.—The chief symptoms on which the diagnosis of disease of the vagus, in any given case, depends, are the laryngeal paralysis, retarded respiration, accelerated or retarded action of the heart, and vomiting. The diagnosis of the seat of the disease rests upon the distribution of the symptoms, and on the associated disturbance. It is especially important to remember that this nerve suffers in polyneuritis. Disease of the trunk of the vagus is much less common than disease of its branches or roots. Paralysis of one vocal cord, for instance, is almost always the result of pressure, either on the recurrent laryngeal or on the roots of the spinal accessory at the medulla. In the former case there are indications of disease in the thorax; in the latter, other nerves suffer, especially the hypoglossal. Bilateral paralysis of the larynx, if considerable, suggests central disease—degeneration of the nuclei in the medulla; if slight, it may be of local origin and independent of a lesion of the nerve itself, as in the case of the "phonic" adduction-palsy. Disease of both recurrent laryngeals is very rare, but it must be remembered that disease of one recurrent has been known to influence both cords. In most cases of pressure on the nerve the compressing disease causes other obtrusive indications of its presence and position, the chief exception being deeply seated tumours of the thorax.

TREATMENT.—The chief element in treatment is to remove the morbid process by which the damage to the nerve is produced; the measures to be employed must vary according to the nature of the process, and have been already fully described in connection with the other cranial nerves. The frequency with which a lesion of the nerve-roots is due to syphilis must be especially remembered. Central degenerative processes are, for the most part, beyond the influence of drugs; their treatment, as far as it is practicable, is described in the section on bulbar paralysis.

The chief division of the pneumogastric for which special treatment may be necessary is that for the larynx. The causes of organic damage to the recurrent nerves are generally grave progressive diseases, such as cancer or aneurism, beyond the effective range of medicine or surgery. The fact that scrofulous enlargement of lymphatic glands is an occasional cause of compression of the recurrent nerves should be remembered; and cod-liver oil, iodide of potassium, and arsenic should be given whenever it is probable or even possible that the disease is of this nature. Moreover, here also syphilis may be at work.

In paralysis secondary to inflammation of the larynx or to cold, the appropriate treatment for the laryngeal disease should be combined with stimulating applications or blisters to the exterior of the larynx. The insufflation of strychnine has been recommended, but the amount absorbed is uncertain and variable, and dangerous symptoms have ensued. It is probable that all the good which strychnia can accomplish is to be obtained from its administration by the stomach or the skin. Hypodermic injections are unquestionably useful in laryngeal palsy; gr. $\frac{1}{60}$—$\frac{1}{30}$ of the nitrate may be injected daily. It is of especial value in hysterical paralysis, in phonic paralysis, and in diphtheritic palsy.

Electricity has been used in various ways. (1) One electrode has been introduced into the larynx and placed near the paralysed muscle, with the guidance of the laryngoscopic mirror, the other electrode being placed outside the larynx. (2) A double laryngeal electrode has been used, with two small rheophores close together. (3) One electrode has been placed in the pharynx at the back of the larynx, the other outside. (4) Both poles have been placed outside the larynx, and the current passed through it. The intra-laryngeal application, even in the most skilful hands, is not pleasant to the patient, and can only be borne for a few seconds at a time. The percutaneous method is painful, although far less so than the other; a stronger current can be borne and for a longer time, and, if the current cannot be localised with the same precision, the muscles can be more effectually stimulated. The positive rheophore may be pressed behind the jaw, and the negative pressed firmly and moved down the side of the larynx and trachea. In thin persons at least, the superior and inferior nerves may be thus directly stimulated. Either faradism or voltaism may be applied externally; in organic disease of the nerve the latter only will influence the muscles. But the value of electricity in organic disease of the nerves is probably not great. In functional palsy, phonic paralysis, and the like, the nerves not being degenerated, faradism can be employed, and is often distinctly useful. It is highly probable that the chief results obtained by electricity are due to the stimulation of the sensory nerves, and to the indirect influence thus exerted on the centres.

In the gravest form of laryngeal palsy, paralysis of the postici, the utility of electricity is practically limited to hysterical cases, and even in them it must be employed with some caution. The separate stimulation of the postici can only be effected by the intra-pharyngeal method, the electrode being placed at the back of the cartilage, a little on one side of the middle line. Any other method of application will stimulate the adductors far more than the abductors, and so increase the perilous constriction of the glottis. Even the intra-pharyngeal method is not free from this danger. All stimulation of the sensory nerves has a reflex action chiefly on the adductors, by virtue of the central reflex mechanism for guarding the air-passages against the entrance of foreign bodies. Hence even the intra-pharyngeal application is in danger of doing more harm indirectly than good directly.

In some cases of phonic paralysis, careful laryngeal gymnastics have been found of service, as in making the patient utter or sing certain simple vowel sounds without complicating articulation. Treatment by manipulation of the larynx was proposed some years ago by Oliver,* and has been found useful by Gerhardt and others. It consists in pressing firmly with the thumb and forefinger on each side of the thyroid cartilage, at the upper and hinder part. During the compression the patient is made to utter a simple sound, and then is often able to speak, at first in a weak, and afterwards in a stronger voice. Vocalisation once effected, the patient is often able to continue to speak. Laryngoscopic examination during the compression shows that the manipulation approximates the arytænoid cartilages and the vocal cords, and at the same time makes them tense. The treatment of hysterical aphonia is described in the chapter on that disease.

In the treatment of *laryngeal spasm* the most important thing is the removal of the condition on which it depends, by the treatment of any local laryngeal irritation and of any diathetic state which exalts the irritability of the central nervous system. Bromide of potassium or ammonium at once lessens the irritability, and cocaine applied as a spray has a like action, but these are in most instances palliative only. Useful as they are in removing spasm for a time, it is necessary also to strengthen the nerve-centres by tonics in order to prevent a recurrence. The spasm is usually removed for a time by the inhalation of chloroform, and often by nitrite of amyl. Ten-grain doses of chloral have been recommended by Johnson as a substitute for chloroform, but chloral is, as a rule, inferior to bromide. Spasm due to local laryngeal irritation is often relieved by a necklet of ice; a long narrow tube is made with gutta-percha tissue, the edges being stuck together by means of chloroform, and this is filled with small pieces of ice and placed round the neck.

SPINAL ACCESSORY NERVE.

(EXTERNAL PART.)

Of the two parts of which the spinal accessory nerve consists, the "accessory" part (which arises from the medulla oblongata, and, joining the pneumogastric, supplies the laryngeal muscles) has been already described. The "spinal" portion, as it is termed from its origin, or "external portion," as it is sometimes called from its distribution, is virtually a series of fibres of the motor cervical nerves, that have the unusual course of ascending to the cranial cavity and leaving it again with one of the cranial nerves to be distributed to the cervical muscles. The root-fibres arise from the middle of the lateral column of the cord, but the fibres pass through this, and spring from the nerve-cells of the anterior cornu, just as do the anterior roots of the cervical nerves. As the fibres have the same origin as the motor cervical nerves, so they have the same distribution—to two cervical muscles, the sterno-mastoid and trapezius, which are supplied in part by this nerve and in part by the cervical nerves. The nerve usually perforates the sterno-mastoid, and supplies this muscle almost entirely, the other nerves to it being unimportant branches from the second and third cervical pairs. The fibres can be traced almost down to the lower border of the trapezius, but this muscle receives a larger supply from the cervical and upper dorsal nerves. Only paralysis of the nerve is here described. The muscles supplied by it are frequently the seat of spasm, which produces the condition known as "torticollis." But the spasm often passes beyond the distribution of this nerve, and is therefore more conveniently considered, in connection with other forms of spasm, at a later page.

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* 'American Journal of Med. Science,' April, 1870, p. 305.

Causes.—The nuclear grey matter from which the nerve arises may participate in central degeneration, causing wasting in these muscles, associated with more extensive muscular atrophy (see vol. i, p. 483). The nerve may be damaged in the neighbourhood of the foramen magnum by local meningitis and compression, and both nerves may be thus affected. At the side of the medulla, moreover, the external part may suffer with the hypoglossal, and with the fibres for the larynx. Outside the skull it may be damaged by wounds, by deep-seated tumours, by caries of the higher cervical vertebræ, and by abscesses springing from the cervical glands, and sometimes, although rarely, by rheumatic neuritis.

Symptoms.—The effect of disease of the nerve is paralysis of the muscles supplied by it. If the trunk of the nerve is diseased, the sterno-mastoid suffers much more than the trapezius, and the latter chiefly in its higher parts. Wasting almost always accompanies the loss of power. The paralysis of the sterno-mastoid is shown by an absence of the normal prominence of the muscle in movements of the head, and by defective power of rotation of the head to the side opposite to the paralysis. Paralysis of one sterno-mastoid does not cause any deviation of the head when at rest, but such deviation may arise from secondary contraction of the unopposed muscle on the other side. There is no such thing as a pure paralytic torticollis.

The only part of the trapezius that is completely paralysed by disease of the spinal accessory nerve is the highest portion, which descends from the occipital bone to the acromion. Instead of the nearly straight contour which this muscle gives to the outer side of the neck in the normal condition, the neck presents a concave curve, and the difference between the two sides is brought out strongly by a deep inspiration, the action in which this muscle is chiefly employed. The weakening of the second part of the trapezius allows the shoulder to fall a little; the scapula recedes from the spine, and is rotated, the lower angle inwards, in consequence of the unopposed action of the rhomboids and the levator anguli scapulæ. Elevation of the arm is also impaired, because the deltoid has lost some of the support from which it acts. But the middle part of the trapezius is never completely paralysed from disease limited to the accessory nerve, by reason of the additional innervation from the spinal nerves.

In bilateral paralysis of these muscles the power of supporting the head in the upright posture is impaired. If both sterno-mastoids are affected, the head tends to fall backwards; if both trapezii, it readily sinks forwards, so that the chin rests on the sternum. Such defective power of support of the head is not uncommon in children in consequence of chronic meningitis about the foramen magnum, damaging both spinal accessory nerves; and it is conspicuous in many cases of progressive muscular atrophy (vol. i, fig. 136). In recent cases of injury to the nerve the muscles present the characteristic reaction of nerve degeneration. In central disease the reaction varies, as it does elsewhere in progressive muscular atrophy.

The distribution of the symptoms differs according to the seat of the disease. When this is in the central grey matter other muscles are always involved, and the distribution of the paralysis in the region of the spinal accessory varies much. As a rule, the highest part of the trapezius suffers later than the rest of the muscle, and for this reason Duchenne called it the *ultimum moriens*. But this rule is not invariable; I have known this part to be the first to suffer. In disease of the trunk of the nerve all parts are involved. Not infrequently the nerve is damaged, by local disease or injury, after it has passed through the sterno-mastoid, and then this muscle escapes, and the trapezius alone suffers. When the lesion involves the intracranial part of the nerve, it is common for the internal or accessory portion to be likewise affected, and there is then paralysis of the vocal cord on the same side. If the lesion is within the skull the hypoglossal is frequently damaged also, and sometimes the palate.

TREATMENT.—The treatment of paralysis of the external part of the spinal accessory is, first, that of the morbid process by which it has been damaged; and secondly, the stimulation of the paralysed muscles by electricity for the purpose of maintaining or improving their nutrition during the recovery of the nerve. The form must be used to which the muscles most readily respond.

HYPOGLOSSAL NERVE.

The hypoglossal nerve is the motor nerve for the tongue, for the depressors of the hyoid bone, and for some of the elevators (hyoglossus and genio-hyoid); it arises from the medulla oblongata beside the olivary body. Its origin has been already described (p. 44). The fibres within the skull are close to those of the spinal accessory and pneumogastric, a relation that is resumed for a short distance in the upper part of the neck, after the passage of the nerve through a separate foramen in the occipital bone. The nerve, however, soon leaves its deep position to course forward to its distribution. Its most important connection is with the petrous ganglion of the pneumogastric, and with the loop joining the first two cervical nerves, from which, and not from the hypoglossal nucleus, the fibres are apparently derived which supply the depressors of the hyoid bone (Horsley and Beevor).

PARALYSIS.

CAUSES.—(1) Nuclear disease which is usually degeneration, rarely sudden softening from vascular occlusion, or an acute process from inflammation. It forms part of bulbar paralysis, acute and chronic, and has occurred in rare cases of locomotor ataxy. The affection is almost always bilateral, the two nuclei being so near together that even an acute lesion scarcely ever affects one only, although this has been observed when the onset was sudden,* and very rarely in chronic degeneration, as in tabes† and general paralysis. The lips and other parts almost always suffer as well. (2) Supra-nuclear disease, involving the motor tract anywhere between the medulla and the lowest part of the ascending frontal convolution or the root of the third frontal,‡ and of any nature, may paralyse the tongue on the opposite side. (3) Infra-nuclear disease: (*a*) within the medulla, the root-fibres are occasionally damaged by softening or by a tumour, usually in association with the contiguous motor tract of the opposite limbs; (*b*) outside the pons, the fibres of origin are damaged by meningitis, simple or syphilitic,§ and by growths. In thickening of the bones of the skull the nerve may be compressed within its foramen. In its course outside the skull the nerve is occasionally damaged by deep-seated tumours, by local cellulitis, by mischief communicated from caries of the highest cervical vertebræ, or is injured by penetrating wounds. In this position the spinal accessory often suffers with it, and paralysis of the palate, occasionally of the vocal cord, results, with or without some wasting in the trapezius and sterno-mastoid. The hypoglossal nerve is rarely the seat of isolated neuritis.* Hemiatrophy of the tongue is sometimes associated with that of the face (*q. v.*), but is probably independent of the hypoglossal nerve.

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* Hirt, 'Berl. kl. Wochenschr.,' 1885, No. 26.

† Raymond and Artaud, 'Arch. de Phys.,' No. 3, 1884. The degeneration was proved post mortem. Many similar instances of tabetic palsy have been since observed.

‡ Beneath which a small focus of softening has affected the tongue only (Edinger, 'Deut. med. Wochenschr.,' 1886).

§ A good example of damage by chronic meningitis is recorded by Hayem and Giraudeau ('Rev. de Méd.,' March, 1883).

SYMPTOMS.—The effects of disease of the hypoglossal nerve are motor only; no loss of sensation results. In paralysis of one hypoglossal nerve the tongue at rest is in its normal position in the mouth, but its root is higher on the paralysed than on the unparalysed side, in consequence of the loss of the tonic contraction of the posterior fibres of the hyo-glossus. Within the mouth the movement is deficient towards the paralysed side. When protruded (Fig. 112), however, the tongue deviates towards the affected, and from the unaffected side, because, in protrusion, the tongue is pushed out by the fibres of the genio-glossus and is pushed towards the weaker side. The point of the tongue is often curved towards the affected half. In complete bilateral paralysis the tongue lies motionless within the mouth, and cannot be protruded. Articulation is always impaired in bilateral paralysis, but one-sided palsy, even considerable, interferes very little with speech, chewing, or swallowing. Mastication is hindered because the tongue fails to keep the food between the teeth, and the loss of the propelling power of the tongue may make it difficult for the patient to get the food into the throat. The nutrition of the tongue varies according to the seat of the disease: when above the nucleus, wasting is absent or insignificant; but when the nucleus or nerve-fibres are diseased, the tongue wastes and shrinks. The wasting is of the muscular tissue; the mucous membrane is thrown into conspicuous irregular folds, prominent rugæ with deep furrows between them. Sensation is not impaired, nor is taste except in slight degree in consequence of inability to move sapid substances about in the mouth.

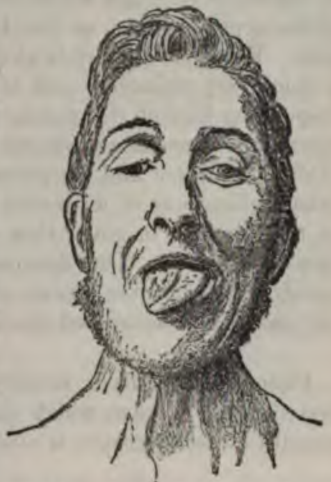

FIG. 112.—Paralysis and wasting of the right half of the tongue, due to disease of the hypoglossal nerve.

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* A case of isolated affection of this nerve in a lad aged thirteen, possibly a rheumatic neuritis, has been recorded by Erb ('Deut. Arch. f. kl. Med.,' xxxvii, p. 265).

DIAGNOSIS.—The position of the lesion is indicated by the associations of the paralysis. If the disease is in the motor tract above the nucleus (pons, crus, or hemisphere), there is almost always hemiplegic weakness on the side of the paralysis of the tongue. In disease of the nucleus the paralysis is commonly bilateral, is associated with paralysis of the lips and throat, and there is usually wasting. Disease of the fibres of origin within the medulla is associated with paralysis of the opposite limbs, so that the tongue deviates from the paralysed side. When the disease is at the surface of the medulla the paralysis is commonly unilateral, and is associated with paralysis of the corresponding half of the palate and vocal cord; and this is also true when the disease is outside the skull, although the associated palsy is often incomplete and irregular (see p. 285). There are commonly other indications of a morbid process in this situation. In disease of the fibres of origin within or outside the medulla there is commonly wasting. The diagnosis of the pathological cause of the paralysis rests on the course of the affection, and on the presence of any causal and associated condition.

PROGNOSIS.—This is usually unfavorable, on account of the character of the disease, which damages the nerve or centre. Even in syphilitic cases recovery is often incomplete.

TREATMENT.—The treatment of paralysis of the hypoglossal nerve is that of the causal disease. Tonics, counter-irritation, iodide of potassium, and mercury, with occasionally the application of electricity to the tongue, are the most important remedies to be employed according to the etiological indication. The most convenient method of applying electricity is by means of a tongue depressor in a wooden handle, the blade being insulated by a coating of sealing-wax where it comes in contact with the lips.

SPASM OF THE TONGUE.

The tongue participates in certain forms of general spasm, such as those of epilepsy and chorea. The tongue-biting in epileptic attacks is due to the organ being jerked between the teeth by the genio-glossus, when the jaws are brought together by the spasm in the masseters. Spasm occurs also in some forms of stuttering. A peculiar affection, allied to stuttering, in which an attempt to speak brought on spasm in the tongue and muscles attached to the hyoid bone, was described by Fleury* under the name "aphthongia." It is especially excited by emotion, and has been compared to writers' cramp.

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* Fleury, 'Gaz. hebd.,' 1865, No. 16. The case (in a man) was complicated by convulsions and other indications of cerebral disturbance. It is possible that the symptoms were of functional origin, as similar spasm certainly was in cases (of

The tongue is a not infrequent seat of spasm in hysteria, in which protrusion may occur during paroxysmal convulsive attacks, or rarely without spasm elsewhere. More frequent in this disease is spasmodic deviation of the tongue when it is put out. I have seen this as an isolated symptom in a child, continuing for some weeks, and also in association with some forms of hysterical spasm about the face. Thus in the case of hysterical ptosis, shown in Fig. 102, the tongue deviated to the left on protrusion, and continued to do so as long as the affection of the eyelids lasted. In another case, in a young woman, similar deviation to the left was associated with persistent tonic spasm, by which the left angle of the mouth and the lower lip were drawn downwards and outward. It is probable that some other cases of spasm of the tongue, recorded as idiopathic, were due to the same disease. A girl,[*] aged nine, suffered for some months from attacks, recurring every ten minutes, and sometimes during sleep, in which the tongue was protruded in tonic spasm, and towards the end of the attack the tip was turned upwards; if the mouth was shut at the time, the tongue was firmly pressed against the teeth. Her own explanation was that she felt an irresistible desire to protrude the tongue. A young man, during two years, had from fifteen to thirty attacks daily of cramp-like protrusion of the tongue, accompanied with pain in the muscles connecting it with the hyoid bone.[†] In another instance of a girl suffering from peculiar hallucinations, whenever the tongue was protruded it presented very rapid movements of protrusion and retraction.[‡] An allied condition probably caused clonic spasm (also in the stylo- and genio-hyoid) which existed during puerperal melancholia.[§]

Spasm in the tongue seems to be sometimes caused by irritation of the fifth nerve. In one instance, it existed during each paroxysm of neuralgia in the lingual nerve.[‖] In another case, dental caries with ulceration of the gums seemed to be the cause of attacks of spasm, in which the tongue was as hard as a piece of wood, and was curved upwards, with the tip toward the palate. The spasm afterwards spread to the face, neck, and arm, and ceased on the removal of the teeth.[¶]

Paroxysmal clonic spasm in the tongue has been occasionally met with, especially as a functional affection, as which it may accompany similar spasm in the face, or may occur alone. Constant clonic spasm on one side, involving also the stylo-glossus and genio-glossus, coincided with puerperal melancholia, and ceased with it, in one recorded

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children) recorded by Panthel ('Deut. Klinik.,' 1855) and Vallin ('Gaz. Hebd.,' 1865, No. 17).

[*] Dochmann, 'Berl. kl. Wochensch.,' 1883, No. 1.

[†] Personali, 'Acad. di Med. di Torino,' liii, 1891.

[‡] Erb, 'Krank. der Periph. Nerv.,' 2 Aufl., 1876. p 296.

[§] Sepelli, 'Riv. Sper.,' 1886.

[‖] Romberg, 'Lehrbuch,' 3 Auf., 1857, p. 388.

[¶] Mitchell, 'Med.-Chir. Trans.,' vol. iv, p. 75.

case.* More frequently the spasm has been paroxysmal, and has accompanied facial spasm (*q. v.*). A man, aged thirty-three, was liable to attacks of protrusion of the tongue, which was pushed out forty or fifty times a minute. Each attack was preceded and accompanied by peculiar sensations in the left side of the tongue and adjacent gums. The spasm seemed to be greatest in the left half of the tongue, and it spread to the left side of the face, where there was slight loss of power. Mastication excited the spasm; speaking did not. Remak, who recorded the case,† thought the spasm was probably of cortical origin, and the contiguity of the facial and lingual centres gives support to the opinion. The attacks ceased under iodide of potassium and electrical treatment. Somewhat similar paroxysms of clonic spasm were limited to the tongue in a case described by Berger.‡ A sense of tension in the organ preceded each attack, which lasted one or two minutes. The attacks occurred during sleep as well as by day, and continued during a year and a half. The patient was a woman aged twenty-eight; she was anæmic, and recovered when treated with iron and change of air. In another case a man, aged forty, had suffered for two years from attacks in which the tongue was pushed out and drawn back more deliberately. The intervals between the attacks varied from a few hours to several weeks.§

It seems clear, from these meagre facts, that spasm in the tongue is generally paroxysmal, and dependent on functional states of the nervous system that are removable by tonic treatment. It thus differs essentially from spasm in the face and neck.

Lingual Dysæsthesia is also met with, sometimes as an obstinate and obtrusive malady, giving rise to the impression that the patient has organic disease of the organ, especially cancer. But the symptom is allied to neuralgia, in connection with which it is described.

PARALYSIS OF THE PALATE.

The movement of the palate that is of chief medical importance is its elevation, which is best seen when the patient utters a long "Ah;" the base of the palate is then drawn up, so that a depression is formed in the middle line of the upper half of the palate. It is probable that this movement is produced chiefly by the levators, each of which spreads out towards the middle line, and there blends with its fellow. If this movement is lost on one side, when the patient utters the sound the middle of the soft palate is drawn a little towards the unaffected side, and a depression is formed on this side of the middle line, instead of in the middle line itself. The difference between the height of the edge of the palate on each side, at rest, is of little significance, since a difference is very common in health: loss of action of one side of the azygos uvulæ is also seldom to be recognised in cases of unquestionable paralysis of the palate. In bilateral palsy, however, the palate hangs lower than normal, and the uvula is long and flaccid. The effect of palsy of the palate is to permit the regurgitation of liquids from the throat into the nose, and to interfere with speech by causing a persistent nasal resonance, and by preventing the compression of air necessary for the articulation of the explosive labial consonants. Paralysis of one side of the palate does not usually cause symptoms, and is only discovered by an examination of the throat.

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* Sepelli, 'Riv. Sper. di Fren.,' 1886.

† 'Berl. klin. Wochenschr.,' 1883, No. 34.

‡ 'Neur. Centralbl.,' 1882, p. 49.

§ Ibid.

The origin of the nerve supply to the palate is one of the most obscure questions connected with the anatomy of the cranial nerves. The levator palati and azygos uvulæ are said to be supplied from the Vidian nerve, and the tensor palati from the otic ganglion of the fifth. The fibres from the Vidian are commonly thought to be derived from the facial nerve, but we have already seen reason to doubt the truth of this opinion. It is certain that the movement of the palate just described, of which the one sided loss is the most definite and frequent, is innervated from one of the nerves that arise from the medulla oblongata. Whenever disease at the surface of the medulla damages the hypoglossal and spinal accessory nerves so as to paralyse the tongue and vocal cord on the same side, it will almost always be found that this movement of the palate is lost.* It is certain, therefore, that it depends on one of these nerves, as was pointed out, from experimental results, by John Reid in 1838, by Bischoff in 1842, and afterwards by Stilling, Wagner, Bernard, and others.† That the innervation is really from the spinal accessory has been conclusively proved by Beevor and Horsley, who found that the palato remained still when the facial or glosso-pharyngeal was stimulated within the skull, but moved when the spinal accessory was stimulated.‡ Fibres from this nerve may pass to the palate by the branch that the pneumogastric gives to the pharyngeal plexus. In some cases of this paralysis the external muscles supplied by the spinal accessory are wasted. The same combination of palsy of tongue, palate, and larynx occurs also from disease within the medulla, damaging the bulbar nuclei, and is considered in another section. The causation and treatment of unilateral paralysis of the palate are the same as of disease of the tongue, already described.

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* The frequency of this fact was first pointed out by Hughlings Jackson ('London Hosp. Rep.,' vol. i, 1864).

† See an instructive paper by W. A. Turner, 'Journal of Anat. and Phys.,' 1889.

‡ 'Proc. Royal Society.' That the spinal accessory is the nerve for this movement of the palate was suggested by S. Mackenzie ('British Med. Journal,' March 3rd, 1883), and also in the first edition of this work.

LOCALISATION OF CEREBRAL DISEASE.

(RELATION OF LOCALITY TO SYMPTOMS.)

The chief facts regarding the relation of symptoms to locality of lesion have been already incidently described in the account of the functions of the brain and of the symptoms themselves. Reference must be made to this when the object is to ascertain the position of a lesion from the symptoms it produces. It is convenient, however, to recapitulate the various effects, regarded from the side of locality, that are caused by disease in the several parts of the brain, so far as these are known, and are of importance in diagnosis. The variations that depend upon the nature of the lesion will be described in the account of the special diseases. The chief precautions that have to be observed in drawing conclusions from observed facts have been mentioned on p. 15.* It should be remembered that the symptoms, and their combination constitute the evidence as to the seat of the disease. The mode of onset and course indicate its nature, which is only of localising significance in so far as some lesions are more common than others in certain parts.

Cerebral Cortex.—It is convenient to consider *seriatim* the several regions into which the cortex is commonly divided, beginning with that in which disease most frequently causes conspicuous symptoms.

Central Region, i. e. the ascending frontal and parietal, anterior two thirds of superior parietal lobule, and paracentral lobule on the inner surface. Destruction causes hemiplegia on the opposite side, permanent, with secondary degeneration of the pyramidal tract, and rigidity of the limbs; a partial lesion affects face, arm, or leg, according to its position (see p. 80). Partial lesions are very common, first, on account of the wide extent of the central region; and secondly, because the region is supplied by different arterial branches, and softening from their occlusion is frequent. Partial palsy, "monoplegia," is far more common from disease of the cortex than from disease more deeply seated; hence it always *suggests* a cortical lesion, but does not *prove* it, because it may be due to a deeper lesion. The leg is probably affected alone only in disease of the medial cortex (paracentral lobule), or when disease of the outer surface is close to the longitudinal fissure. Disease between the longitudinal fissure and the level of the lower frontal sulcus, paralyses the arm,* which is affected alone if the lesion is limited to the middle third of the convolutions (Fig. 113). Probably disease at the junction of the ascending frontal and highest frontal paralyses the foot more than the rest of the leg, and a lesion in the highest part of the arm-region affects the shoulder more than the arm. In drawing conclusions from the preponderant palsy of the extremity of either limb, great care is necessary, because this part is less represented in the other hemisphere than are the muscles of the upper parts of the limbs, and the latter recover some power by compensation (see p. 80). Disease of the ascending frontal, opposite the upper half of the inferior frontal, causes paralysis of the lower part of the face, except the lips, which suffer, with the tongue, in disease of the lowest part of the ascending frontal (Fig. 114). But the bilateral representation of the lips renders the effect of one-sided disease inconspicuous. Disease in the same region sometimes causes transient weakness of the opposite masseter; that of the lowest part, in front, paralyses the opposite vocal cord (see p. 14). On the whole, the paralysing effects of limited lesions of the central region correspond very closely in different cases. Apparent exceptions are sometimes due to the depth to which the disease penetrates. Lesions are seldom confined to the grey substance, and if they extend deeply into the white substance they may interrupt the fibres from another region of the cortex that is not diseased.

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* The most important writings on the subject of cerebral localisations are by Nothnagel, in his admirable and exhaustive 'Topische Diagnostik der Gehirnkr.,' 1879. Ferrier, 'Gulstonian Lectures on Localisation of Cerebral Disease,' London, 2nd ed.; Charcot and Pitres, 'Revue de Méd.,' 1877, 1878, 1879, and 1883, and Allen Starr, numerous collections of facts published chiefly in the 'Amer. Journ. of Med. Science' since 1884.

FIG. 113.

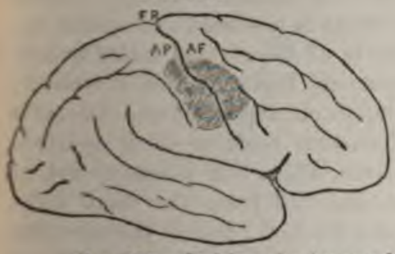

FIG. 114.

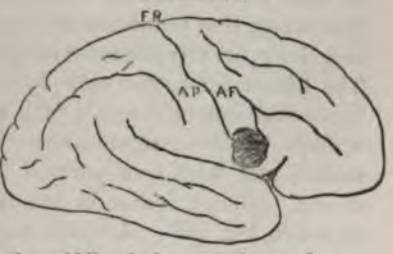

FIG. 113.—Position of a tumour in the middle of the central convolution which caused convulsions beginning in the left arm, and afterwards paralysis of the arm without implication of leg or face. (Müller, 'Trans. Int. Med. Congress,' London, 1881.)

FIG. 114.—Position of a small hæmorrhage which caused paralysis of the tongue and lower part of the face. (Ballet, 'Prog. Med.,' 1880, p. 762.)

Irritating disease in the central cortex, and also stationary partial lesions cause convulsion, which begins locally in the leg, arm, or face, as the disease is in or near the region destruction of which causes palsy of the part. Thus disease of the middle third of the central convolutions often causes convulsions beginning in the hand. According to its degree, the resulting convulsion may be confined to the part whose centre is irritated, or may spread through the whole side, or may be bilateral; the second side being affected after the first (see p. 90). This local commencement is the great characteristic of convulsions from cortical disease. Rarely a lesion elsewhere, either an acute lesion or chronic irritating disease, such as tumour, causes such convulsions. But a stationary lesion, of acute onset, never causes recurring convulsions unless it is in the cortex. Hence initial convulsions of this character suggest cortical disease, but recurring convulsions from a stationary lesion in adult life prove it. They may follow a lesion of the internal capsule in childhood. Such convulsions are thus of great general significance, but the indication they afford as to the exact seat of the disease is not always precise; it is less precise than is paralysis, because cortical disease causing local palsy must involve the centre concerned, but such disease causing local convulsion may be only near the centre. The convulsion may begin by a sensation or by spasm, the significant indication is the part in which the discharge commences, irrespective of its nature. Thus, if the fit begins with tingling in the foot, and the sensation seems to pass up the leg and side, and down the arm, and then the hand begins to twitch in spasm, the phenomena indicate disease in or near the leg centre, as certainly as if it began by motor spasm. Further, certain centres seem to be more readily discharged than others, and an influence that exalts irritability in a considerable area may be first manifested in these centres, not because the morbid state is greater in them than elsewhere, but because they respond to it more readily. The centre that influences the movement of the head and eyes to one side, is apparently the most sensitive of all the cortical motor centres. The fits of idiopathic epilepsy commonly begin by this movement; hence such commencement of a convulsion due to organic disease is of little localising significance. Persistent tonic or clonic spasm is seldom met with from cortical lesions, but a few instances are on record. Clonic spasm in the face resulted from the small lesion shown in Fig. 107. A lesion beneath the lower part of the ascending frontal caused trismus during the two days the patient lived,* and convulsive spasm in the shoulder muscles was due to a tumour at the root of the second frontal.†

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* Yet the arm has been paralysed by a lesion only a few millimetres from the longitudinal fissure.

Besides the motor palsy, disease of the central cortex often causes loss of the sense of posture in the part paralysed, but as this loss also occurs in disease of the motor path in the internal capsule its diagnostic significance is not great. Cutaneous sensibility may also be impaired by disease of the central region, and the sensory paralysis corresponds in seat to the motor palsy, but it is always very much slighter in degree, and is chiefly marked at the extremity of the affected limb.* It may be absent, and hemianæsthesia never results from disease limited to this part. Tactile sensibility is more impaired than is sensibility to pain. There are commonly no definite mental symptoms.

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* 'Revue de Méd.,' Oct., 1882.

† Putnam, 'New York Med. Assoc.,' 1891.

Prefrontal Lobe.—A lesion at the posterior extremity of the upper frontal convolution, at its junction with the ascending frontal, may cause paralysis of the foot, or convulsion beginning in the foot; but with this exception no permanent motor paralysis results from disease of this region, even when it extends up to the ascending frontal. The extensive lesion shown in Fig. 115 produced no palsy. If the movement of the head and eyes to one side is represented in this region, as is suspected from the results obtained by experiments upon animals, the loss in man is quickly compensated by the other hemisphere, and disappears. Exceptions are, indeed, sometimes seen, in which hemiplegia is accompanied by prolonged deviation of the head and eyes, but the seat of disease causing this has not been ascertained. Unsteadiness on walking, like that of cerebellar disease, has been described as frequent in prefrontal growths.† It is distinguished by associated hemiplegia, and the local headache and tenderness on percussion.‡ Local convulsions are sometimes caused by irritating disease adjacent to the ascending frontal. Disease of the third frontal, on the left side, impairs voluntary speech in the manner already described (p. 114), and a similar effect is produced by disease of the corresponding region on the right side in left-handed persons, and in those who have recovered speech after destruction of the left motor speech region. No sensory symptoms are caused by disease in the prefrontal lobe, but in some cases considerable mental change has been observed, various in character, but sufficiently frequent and considerable to be of significance.§ A very large number of cases are on

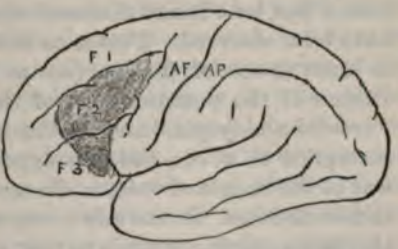

FIG. 115.—Extensive softening of cortex of left prefrontal lobe which caused no motor or sensory symptoms.

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* References to the evidence of this will be found in the chapter on the functions of the cortex. A series of cases of disease of the central region with loss of sensation is recorded by Petrina, 'Prag. Zeitschr. f. Heilk.,' ii, 1887, No. 5.

† Bruns, 'Deut. med. Wochenschr.,' 1892.

‡ The experiments of Ferrier, Mott, and Schäfer suggest that the centre for movement of the head is at the root of the second frontal, and that for the eyes in front of this; that for a downward and lateral movement being in the upper and for an upward and lateral movement in the lower part of the centre. Extirpation of the opposite centres restrict movements (on stimulation) to the opposite eye.

§ A familiar instance is the American "crowbar case," in which a crowbar was driven through the left frontal lobe, from the eye to the coronal suture, and the patient lived for thirteen years, with only a childish mental state and epileptic fits.

record of disease and injury of various kinds in this part, in which psychical disturbance was the only symptom. It is greatest when both frontal lobes are diseased, and sometimes passes away (by compensation) when the lesion is unilateral.

Parietal Lobe.—The symptoms produced by disease of the ascending parietal and the superior parietal lobule have been mentioned already. The posterior extremity of the latter, adjacent to the parieto-occipital fissure has been found diseased when no motor or sensory symptoms have been observed. Ptosis, on the opposite side, has been observed in many cases of disease (see p. 19) of the lower parietal lobule. Disease of the posterior part of the inferior lobule probably causes "crossed amblyopia," and on the left side interferes with the visual perception of words, and may apparently cause "agraphia" from the loss of the images of written characters; if bilateral, it perhaps causes mind-blindness. On one side it may be associated with hemianopia from the disease passing inwards to the "optic radiation." Extensive disease of the whole parietal lobe seems to lessen sensibility in the trunk and limbs on the opposite side. Such an isolated effect has not been observed, but it is certain that disease involving the parietal as well as the central cortex causes greater sensory loss than does a lesion that is confined to the central region.

Occipital Lobe.—Disease of the apical region, and especially of the cuneus, causes lateral hemianopia, and a partial lesion causes loss of part of the half-field, a quadrantic or sectional defect, which, when above, indicates a lesion in the upper part of the cuneus, when below, in the lower part. Lesions of the outer part of the apex have been associated with hemianopia, but the readiness with which the fibres from the cuneus may be reached must be remembered. A lesion of both occipital lobes causes complete loss of sight, and is the probable cause of this when it is sudden in onset.[*] The loss of the second half-fields may be transient, due to inhibition by the the primary lesion. As a sequel to apoplectic coma, it is mentioned later (p. 317). If the onset is deliberate and consciousness retained, the primary and permanent hemianopia may precede the secondary and transient loss.[†]

Possibly disease of the anterior part of this lobe causes colour hemianopia. No motor or other sensory symptoms are known to be produced by disease elsewhere in this lobe. Conjugate deviation of the eyes occurs from the side of the disease, sometimes in irritating lesions, and is doubtless a reflex subserved by the fibres that pass forward to the prefrontal lobe.

Temporo-sphenoidal Lobe.—Only auditory symptoms are caused by disease of the outer aspect: deafness in the opposite ear when the disease involves the hinder half or third of the first, or first and second convolutions, and an auditory aura, if convulsions are caused by a lesion in or near this part. Such an aura, with other symptoms of a stationary or progressive lesion, justifies the diagnosis of disease of this part. A tumour caused convulsions preceded by the sound of bells; in another case of tumour in this situation, recorded by Fergusson,* the diagnosis was curiously complicated by old ostitis on the opposite side. The deafness is not permanent, but if, as in Mills' case, there is a second lesion in the corresponding region of the other hemisphere, total deafness results. Conjugate deviation of the eyes has been observed as in disease of the occipital lobe; it is probably reflex, and due to the auditory impression. The auditory region is said to be more extensive on the left side, and disease of the posterior third of the first temporal causes word-deafness and the "paraphasia" that results from the loss of the leading element in the subjective revival of words, and consequent "verbal amnesia." Disease of the highest (uncinate) convolution on the medial surface, near the apex of the lobe, may disturb the sense of smell on the same side (see p. 20).

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[*] Sudden complete blindness, except perception of a bright light in the outer angle of the left field, in a man of sixty, was found to be due to softening of the whole of the left occipital lobe and of the right angular gyrus, &c., and subjacent white substance, involving the fibres of the "optic radiation" (Sioli, 'Versam. Psych. Vereins Karlsruhe,' November, 1892).

Case of Noyes, 'Journ. Nerv. and Ment. Diseases,' 1889.

Extensive lesions of the cortex around the fissure of Sylvius, extending over a considerable area of the central, parietal, and temporal lobes (such as is shown in Fig. 15, p. 23) may cause hemiplegia and hemianæsthesia involving all the special senses, the opposite eye being the more affected.

Lastly, transient bilateral symptoms may, as we have seen, result from irritative inhibition of the corresponding centres in the opposite hemispheres. It occurs in disease both of the cortex and white substance if so placed as to influence the fibres of the corpus callosum.

WHITE SUBSTANCE OF THE HEMISPHERE; CENTRUM OVALE.—Disease of the white substance causes symptoms resembling, on the one hand, those of disease of the cortex, and on the other of the internal capsule, according as the seat of the lesion approaches one or the other of these structures. Thus a lesion beneath a given part of the central convolutions produces paralysis similar in distribution to that which is caused by disease of the corresponding region of the cortex. Hemiplegia is produced by lesions that interrupt the fibres which converge from the central region to the motor part of the internal capsule. One important difference, however, exists: the local convulsions, that are so frequent and characteristic a symptom of a cortical lesion, are met with in disease of the white substance only when an irritating lesion is situated just beneath the grey substance so as to irritate this directly. General convulsions are rare, and occur chiefly in disease that increases intra-cranial pressure. The loss of speech that results from disease of the fibres passing from the third frontal convolution, to the internal capsule is transient, unless the lesion is just beneath the cortex. Its various characters have been described at p. 114. Anæsthesia occurs chiefly from extensive lesions beneath the parietal and central regions, but in most cases in which considerable hemianæsthesia results from disease of the white substance, this extends down to the neighbourhood of the posterior part of the internal capsule. Hemianopia may be caused by disease of the white substance of the occipital lobe, anywhere between the optic thalamus and the extremities of the hemisphere.

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* 'Journ. of Anat. and Phys.,' 1891.

Corpus Callosum.—The chief lesion that occurs in the corpus callosum is tumour, but this almost always extends into one or both hemispheres. Mental dulness and stupor, with weakness in the limbs, greater on one side, have been present in many cases, and in some there has been disturbance of speech and locomotion; but it is probable that these symptoms (with the possible exception of the mental dulness) are due to the pressure on, or extension into, the cerebral hemispheres. We do not yet know of any symptoms that are the result of the damage to the callosal fibres; it is certain that the symptoms that have been present are indistinguishable from those produced by multiple tumours,* and that their complete interruption (by softening from embolism) has caused no symptoms.†

Central Ganglia and Internal Capsule.—*Internal Capsule.*—Disease limited to the anterior part of the capsule, between the caudate nucleus and the anterior extremity of the lenticular nucleus—so as not to involve the "angle," at the junction of the two parts—is very rare, and we do not yet know whether it causes definite symptoms.‡ Disease of the angle and posterior segment causes hemiplegia of the common type, the lower part of the face, the tongue, the arm, and the leg being all involved, and if the disease is on the left side there may be, at first, defect of speech. The latter is transient; the hemiplegia is permanent if the lesion continues (except only such recovery as may occur by compensation), and the paralysed limbs are the seat of late rigidity. At the onset there may be deviation of the head and eyes, but there is no permanent and complete palsy of any cranial nerve.

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* Some interesting cases of tumour of the corpus callosum have been published by Bristowe, 'Brain,' 1884, p. 315, but the diagnostic indications formulated by him are no exception to the statement in the text. A remarkable case of extensive hæmorrhage into the corpus callosum has been recorded by Erb ('Virch. Arch.,' Bd. xcvii, 329), but the hæmorrhage occurred apparently during the course of cerebro-spinal meningitis, in the symptoms of which any effects of the callosal hæmorrhage were lost. The history hardly seems to warrant the conclusion that no symptoms were produced.

† Kauffmann, 'Arch. f. Psych.,' xviii.

‡ Raymond and Artaud believe that the speech-path is in the anterior segment ('Arch. de Neurologie,' 1884), but it is doubtful whether this path is separate from that for the tongue and lips in the angle of the capsule

Convulsions may attend the onset of an acute lesion, but are infrequent, and, as in other parts except the cortex, they do not recur after the disease has reached a stationary stage. The palsy is thus the typical, common form, the details of which have been described at p. 75. Most cases of simple hemiplegia are due to disease of this part, and we must therefore assume that this region is diseased, unless there are other symptoms to indicate a different seat of the disease. But hemiplegia from disease of the capsule is not always complete; we have seen (p. 29) that the path for the face and tongue seems to pass chiefly at the angle, that for the arm in the anterior, and that for the leg in the middle third of the posterior limb. Small lesions may therefore affect *chiefly* one or the other of these parts; there is seldom an actual limitation of the palsy, although isolated facial hemiplegia has been observed from disease at the bend, and a bilateral lesion further back has apparently caused phonic laryngeal palsy.* Hemianæsthesia may accompany the hemiplegia, from implication of the separate sensory path in the hinder third; such sensory loss sometimes accompanies hemiplegic weakness in which the leg suffers most, a fact which is explained by the contiguity of the fibres for the leg and the sensory path. The hemianæsthesia produced by a considerable lesion of the path in the capsule may be complete, and involve the special senses as well as the skin and the muscles; the loss of vision is hemianopia, as before explained. Smell may also be lost on the anæsthetic side. This region is the "sensory crossway" of Charcot. But the special senses may escape, even when the cutaneous loss is great. At the deliberate onset of an acute lesion, tingling and other sensations are far more common than they are when the disease is limited to the motor part of the capsule. If the hemianæsthesia is partial, from incomplete disease, pains are often felt in the affected limbs, sometimes most persistent from the irritation of the slightly damaged sensory fibres of the internal capsule, while if the lesion is so placed as to influence in like manner the motor fibres, there may be permanent rigidity of the limbs of the opposite side.

Corpus Striatum.—Acute lesions of either caudate or lenticular nucleus generally cause hemiplegia, but this is permanent only if the internal capsule is directly damaged. If the lesion is confined to the grey substance of either nucleus, there are usually no persistent symptoms, motor or sensory. If the lesion is small, and is at a distance from the white fibres of the capsule, there may even be no initial hemiplegia. I have seen a narrow vertical band of central softening, extending from the anterior to the posterior extremity of the lenticular nucleus, when no trace of hemiplegia could be detected before death, and a careful history had elicited no account of any previous paralysis. Even a quite recent hæmorrhage into the lenticular nucleus has caused no symptoms. Chronic lesions, such as tumour, often develop in the grey nuclei without causing paralysis. From these facts it is probable that no sensory or motor palsy is produced by disease of the grey matter, and that the symptoms which occur at the onset of acute lesions are due to the interference with the fibres of the internal capsule, which have been already described. In a few cases[*] mobile spasm (choreoid movement) has been observed when the lesion involved the posterior part of the lenticular nucleus, but in most cases the internal capsule was also damaged. Disease of the grey matter of this or the thalamus has been associated with brief increased temperature (Hale-White) both in experiments and also as a result of disease in man.

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* Eisenlohr.

Optic Thalamus.—Slight hemiplegia has been observed when the disease has been in the middle third of the thalamus, but is absent if the lesion is small or near the ventricular surface; hence there is a strong presumption that the loss of power is due to interference with the motor part of the internal capsule, which is adjacent to the middle third. In disease of this part of the thalamus another motor symptom has been occasionally observed, mobile spasm and incoordination on the opposite side, chiefly in the hand. The symptom usually succeeds transient hemiplegia. In one such case (in an adult) I found a small cicatrix of old softening, limited to the thalamus,[†] and several other similar cases are on record. In one there was softening in the lower part of the thalamus, adjacent to the crus.[‡] It seems probable that the initial hemiplegia is due to the interference with the capsular fibres, and that the subsequent spasm is in some way the direct result of the disease of the thalamus. Disturbance of facial expression has been ascribed to a lesion in the posterior part of each thalamus.[§] Sensation is not impaired by lesions limited to the thalamus. A special relation to the impulses from the muscles has been suggested,[||] but has not received the confirmation reasonably to be expected, although it would agree with many facts and hypothetical explanations of symptoms, as athetosis, cerebellar disease, &c. Hemianæsthesia occurs only when the disease extends into the internal capsule outside the pulvinar. In such cases there may also be hemianopia, and this symptom may, apparently be caused by disease limited to the posterior part of the thalamus itself. I have seen two cases in which there was no other lesion that could be associated with the hemianopia which existed during life, and the pulvinar has been found atrophied with the occipital lobe (and also the anterior corpus quadrigeminum, the external corpus geniculatum, and the optic tract),[¶] and partial degeneration of the optic tract has been traced to it (Noyes).

It has been thought that muscular sensibility is specially impaired by thalamic lesions (Meynert), but the evidence does not satisfactorily exclude the adjacent capsule. Diminution of reflex action has been thought to be another effect, but it is met with in disease of other parts of the brain. Vaso-motor symptoms do not seem to result.

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[*] Demange, 'Revue de Méd.,' May, 1883.

[†] 'Med.-Chir. Trans.,' 1876, p. 318.

[‡] Greiff, 'Arch. f. Psych.,' Bd. xiv, p. 598. Choreoid movements set in suddenly, with transient hemiplegia and hyperæsthesia.

[§] Eisenlohr.

[||] From the symptoms of a tumour (Kiritzew, 'Neur. Cent.,' 1891).

[¶] Eisenlohr, 1890; Stauffer, 1890.

CORPORA QUADRIGEMINA.—Lesions whose influence is confined to the corpora quadrigemina are so rare that there is considerable doubt as to the symptoms produced. It has been thought that disease of the anterior pair causes loss of sight and loss of action of the pupils, but in most of the cases on which the opinion is based the disease was a tumour, and optic neuritis co-existed.[*] Ataxy of movement has been present in some cases of disease of the posterior tubercles, but it resembled closely that due to disease of the middle lobe of the cerebellum, implicatian of which (or of fibres passing from the cerebellum to the brain) may possibly have been the cause of the symptoms. Loss of the upward movement of the eyes was produced, in one case under my care, by a small growth at the junction of the hinder tubercles with the valve of Vieussens, and experiment also suggests a connection between the grey matter of these with this movement of the eyes. But in most cases of disease of the tubercula, causing palsy of the ocular movements, the lesion has extended so deeply as to involve the nuclei of the third nerves. A combination of the above symptoms, however, justifies a suspicion of disease of these structures. If acute, contraction of the pupils showing irritation of these centres adds to the probability of the lesion.

CRUS CEREBRI.—The characteristic symptom is hemiplegia, involving the lower part of the face as well as the limbs, and accompanied by palsy of the third nerve on the opposite side, *i. e.* on the side of the lesion, the two symptoms coming on at the same time. The affection of the third nerve is usually complete, but now and then is partial; sometimes the fibres for the internal ocular muscles have escaped. Convulsions are seldom met with, but some spasm has been observed in cases of tumour. Loss of sensation accompanies the motor palsy when the lesion extends into the upper region of the crus, and is sometimes accompanied by tingling and other signs of sensory irritation. Vaso-motor disturbance is occasionally conspicuous in the paralysed limbs. Hemianopia is much less common than might be expected from the contiguity of the optic tract, and has been met with chiefly in cases of tumour.

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[*] One case of acute lesion causing these symptoms has been recorded by Dr. Bastian, and I have seen one similar case. Hæmorrhage in both occipital lobes is the most likely cause. Bilateral lesions are sometimes simultaneous and symmetrical; probably that on one side can disturb the circulation on the other. A woman was unconscious for several days, evidently from cerebral hæmorrhage. On regaining consciousness she was quite blind. In a few days more vision returned in the lateral half-fields on the left side, but remained permanently absent in them on the right. It would seem that a hæmorrhage in the left occipital lobe had inhibited for a time the corresponding half-vision centres on the other hemisphere. Such an effect may readily be attended by disturbance of the circulation, facilitating another hæmorrhage. Atheroma also is sometimes symmetrical.

Partial lesions in or near the middle line, beneath the corpora quadrigemina, may cause the symptoms of acute nuclear ophthalmoplegia described at p. 198. The paralysis is usually incomplete, variable in distribution, and tends to lessen. A very superficial lesion may cause no hemiplegia, but partial palsy of one third nerve, from damage to the root-fibres, with persistent giddiness supposed to be due to interference with fibres from the cerebellum.

PONS.—Unilateral lesions of the pons often cause palsy of the limbs on one side and of the fifth, facial, or sixth nerve on the other, the "alternate hemiplegia" described at p. 81. But a lesion may be so placed, generally in the upper part of the pons, that the cranial nerves and nuclei escape, and the hemiplegia is then indistinguishable from that which results from disease of the internal capsule. An important unilateral symptom is the loss of the conjugate movement of both eyes towards the side of the lesion, described at p. 186, or corresponding conjugate spasm (often with nystagmus) when the lesion irritates the centres. Bilateral lesions are not uncommon; the limbs or face on both sides may then be involved. Difficulty in swallowing and in articulation is common, but is less pronounced and prolonged than in lesions of the medulla oblongata. The fibres for articulation seem to run in the medial-posterior part of the pyramidal tract, and through those on either side the muscles of both sides can be influenced. Hence a unilateral lesion in the upper part of the pons does not affect articulation, but a bilateral lesion often impairs it extremely. Occasionally disease of the pons causes a defect curiously like that of some cases of motor aphasia, unmeaning syllables being correctly uttered, as "sej-je-pel" instead of the desired word.* Convulsions are very rare in chronic, but may occur at the onset of acute lesions; they have often a peculiar character, both arms or both legs may be chiefly involved, or there may be semi-convulsive paroxysms of coughing. Sometimes there is general rigidity of the limbs, continuous or paroxysmal. The muscles supplied by the fifth nerve may be fixed by spasm causing trismus. Choreoid movements have been noted in rare cases; in one instance there was rhythmical spasm on voluntary movement (Bastian). Loss of sensibility in the limbs and trunk is sometimes associated with the motor palsy, in consequence of the implication of the tegmental region of the pons, and loss of muscular sensibility with ataxy in the arm, has followed a lesion between the hypoglossal roots and the middle line.† The region of the skin &c., supplied by the fifth nerve usually escapes on the side of the hemiplegia, but is anæsthetic, and is sometimes the seat of pain on the side of a lesion, so placed as to involve the nucleus or root-fibres. The motor part of the fifth may suffer without the sensory portion; loss of taste may accompany the palsy. Ophthalmitis is also met with, but much less frequently than when the disease involves the trunk of the nerve. Acute irritating disease, especially hæmorrhage, may cause strong contraction of the pupils, and a lesion that extends into the upper part of the pons, beneath the corpora quadrigemina, may paralyse the external ocular muscles. Auditory symptoms are rare, but deafness has existed on the side of the lesion, or a subjective sensation of sound has attended the onset of an acute process. Giddiness occurs chiefly when the disease is in the neighbourhood of the middle peduncle of the pons; it may be most intense and even amount to actual movement. It is, of course, attended by vomiting, which, without vertigo, is not specially frequent. Disturbance of respiration and of the action of the heart (even to 108°), glycosuria, and albuminuria are sometimes observed, but the urine is seldom altered in cases of tumour. Hyperpyrexia is not uncommon immediately after the onset of an acute lesion of the pons, especially hæmorrhage, and is of grave significance.

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* It may be that the path from the motor speech-centre is distinct from that for mere articulation. Several similar cases are on record in which there was no indication of any disease above the pons.

† Interrupting fibres supposed to ascend to the quadrigeminal fillet (Goldscheider, 'Charité Ann.,' 1891).

MEDULLA OBLONGATA.—Severe acute lesions of the medulla are quickly fatal by interference with the respiratory or cardiac centres, and, on account of the small size of the medulla, and the close proximity of its centres, a hæmorrhage usually has this effect even when of small size. The chief acute lesion that may give rise to a diagnostic problem, is softening from vascular obstruction. Motor symptoms in the limbs are often bilateral, and sometimes involve especially the upper or lower limbs; if one sided, the tongue may be paralysed on the side opposite to the lesion, if this is in the anterior part of the medulla, so as to involve the pyramid and the root-fibres of the hypoglossal nerve. The face (with the exception of the lips) always escapes. The most characteristic symptoms are those that are due to the interference with the bulbar nerves, the hypoglossal, glosso-pharyngeal, and spinal accessory. Such symptoms are seldom unilateral, because they usually depend on interference with the nuclei, and these lie for the most part near the middle line, and suffer on both sides. Hence the resulting palsy commonly involves the orbicularis oris (which is supplied from nerve-cells near the hypoglossal nucleus), the tongue, palate, pharynx, and sometimes the larynx, with impairment of articulation and of deglutition. The paralysis often amounts to almost complete loss of articulation, but loss of power in the pharynx is seldom absolute, perhaps because the glosso-pharyngeal nucleus is farther from the middle line than are those of the hypoglossal and spinal accessory nerves. The resulting symptoms are described more fully in a separate section on the bulbar paralysis that results from nuclear degeneration and acute lesions of the medulla.

Bilateral Lesions in the motor region cause paralysis on both sides, and if the disease is situated in the centres for the muscles of bilateral use, which recover, by compensation, when one hemisphere is diseased, the paralysis of these muscles is great in degree; in completeness it resembles that caused by disease lower down the motor path where the two paths are near together. Thus disease of the highest part of the central regions, which sometimes results from meningeal hæmorrhage during birth, causes palsy of the legs closely resembling that due to disease of the spinal cord. Disease of the lowest part of each ascending frontal convolution causes bilateral palsy of the lips, tongue, and throat, closely resembling labio-glossal paralysis,—"pseudo-bulbar paralysis," it is termed. Soo too in the sensory regions in which compensation occurs; complete deafness may be caused by disease of each first temporal convolution. In the few cases on record in which the symptoms, commonly due to disease of both hemispheres, are produced by disease on one side, it is probable that there is a congenital or old-standing defect on the other side, so that the apparent exceptions do not really contravene the common rule. Bilateral disease of the occipital lobes may cause absolute blindness, a compound of double hemianopia; it has usually developed in two separate attacks, but need not necessarily do so. As the case mentioned on p. 317 (foot-note) shows, it is often needful, when there are bilateral symptoms, to wait a few days in order to exclude inhibition of the centres on the second side.

Cerebellum.—Disease of the middle lobe of the cerebellum causes unsteadiness of movement,—a reeling gait and often a difficulty in standing, a tendency to sway which renders difficult the maintenance of equilibrium. Sometimes, but not often, there is a tendency to fall backwards. There is not the irregular movement of the legs that characterises locomotor ataxy, although in some cases of tabes the unsteadiness closely resembles that of cerebellar disease. In walking, the tendency to oscillation may give a zigzag direction to the walk, and the resemblance to the gait of a drunken person is often close. The arms are usually steady; rarely they present some jerky inco-ordination. The unsteadiness is due to damage to the middle lobe, either from disease in it, or from compressing disease in one hemisphere. If a lesion in one hemisphere does not compress the middle lobe, this symptom is absent, and such a lesion seems *per se* to cause no symptoms by which it can be recognised. Other symptoms of cerebellar disease occur also in many different intracranial maladies, but are more frequent in disease of the cerebellum than in disease elsewhere, so that they have a slight localising value; such are giddiness and vomiting, but these occur only in active diseases that irritate or compress, *e. g.* in tumour and at the onset of acute vascular lesions. The unsteadiness is not necessarily related to vertigo; the two are sometimes associated, but the unsteadiness may be extreme when there is no subjective sense of giddiness. Nystagmus is frequent in cases of tumour, but we do not know how it is caused.[*] In some cases of tumour of the middle lobe the knee-jerk cannot be obtained. Its loss is often not constant; a slight movement may be elicited at times, and at other times none. The loss has been found only on the side opposite to a tumour in one cerebellar hemisphere.[†] There is no evidence regarding the mechanism of this curious symptom, but we may note the contrast it presents to the effect of disease of the motor region of the cerebral cortex, which augments the knee-jerk. The influence, the loss of which leads to increase, may, unrestrained, have the opposite effect, and cerebellar disease may involve the loss of such a controlling and guiding upward influence (see p. 59).[‡] A sudden irritating cortical lesion may abolish the knee-jerk for a few hours.

Many other symptoms are produced by cerebellar disease that exerts pressure. The pyramidal tracts often suffer thus, and hence the limbs are weakened, and the weakness is accompanied by the usual increase of myotatic irritability. Thus in one case of cerebellar tumour there may be no knee-jerk, and in another it may be increased. The cranial nerves, especially the sixth nerves, may likewise be affected by distant pressure. Occasionally there are other signs of compression of the medulla oblongata, such as difficulty of articulation, &c. These symptoms, which are almost confined to morbid growths, are described more fully in the chapter on intra-cranial tumours.

Convulsions are rare in disease of the cerebellum, but have occurred occasionally, epileptiform in type, and probably indirect in origin. Tetanoid rigidity, with bending back of the head, constant or paroxysmal, has been met with, and has been ascribed to the cerebellar disease (Hughlings Jackson). But it occurs only in tumour, and is met with also in cases of infiltrating glioma of the pons, so that it may be pressure effect. Hæmorrhage into the cerebellum sometimes extends into the side of the pons, and the symptoms become indistinguishable from those of an acute lesion in the latter situation.

CEREBELLAR PEDUNCLES.—The superior and inferior peduncles are scarcely ever the seat of isolated disease. The middle peduncle is sometimes separately damaged, but the lesion often involves also either the side of the pons or the adjacent cerebellar hemisphere. The chief symptom is vertigo, a sensation of movement and actual movement, but this is present only in irritating lesions. The sensation is often most intense; the movement may be merely an involuntary motion of the head and eyes, or there may be a strong tendency to an actual rotation of the trunk on its long axis, a forced movement, sometimes present whether the patient is lying or standing, sometimes chiefly marked in the upright posture, when it may cause the patient to turn round and round. Occasionally the eyes are in an abnormal position, one being on a different level from the other. Hemiplegia on the same side is said to occur in rare cases.* With these symptoms there is often evidence of damage to the side of the pons, such as symptoms of paralysis of the fifth nerve, with or without hemiplegic weakness. A stationary lesion may cause no symptoms.

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[*] It was in a case of tumour of the cerebellum that the curious rhythmical spasm in the pharynx mentioned on p. 208 was observed.

[†] Knapp. 'Journ. Nerv. and Ment. Dis.,' 1892.

[‡] The crossed relation of the symptom to its cause, just mentioned, is a difficulty, but we need to know whether it is constant, and also more of the upward connection of the cerebellum. If the above hypothesis is correct, the fibres through which the cerebellum exerts its upward influence may not pass directly to the motor cortex, and a tract of fibres is said to pass by the superior cerebellar peduncle to the optic thalamus of the same side (Marchi). The knee-jerk is sometimes lost on both sides when a tumour is in one cerebellar hemisphere.

VENTRICLES.—The chief disease of the ventricles that gives rise to localising symptoms is hæmorrhage, in the account of which these symptoms are considered. Of the diseases that affect the base, the local diagnosis in hæmorrhage and inflammation is subordinate to that of the morbid process, and the indications of the position of morbid growths are considered in the chapter on intra-cranial tumours. These may occupy one of the ventricles, as the third or fourth, but grow into it from a neighbouring part of the brain or from the fornix. So far as any symptoms can be referred to the ventricular growth, those attending a tumour in the third ventricle resemble the effects of a growth in the corpus callosum, and in the fourth ventricle a tumour of the pons or medulla.

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* Brown-Séquard, Hughlings Jackson.

DISEASES OF THE MEMBRANES OF THE BRAIN.

CONGESTION.

Active congestion of the membranes of the brain constitutes the first stage of inflammation, and has been supposed to exist as an independent condition in some cases, chiefly in children, in which there are acute cerebral symptoms—headache, delirium, convulsions, coma—ending sometimes in recovery, sometimes in death. In the latter cases indications of meningeal hyperæmia have been found. It is assumed that in all these cases the condition is one of active congestion of the membranes. In fatal cases the symptoms are similar to those of rapid meningitis, and it is probable that the condition is of this nature. In other cases it is possible that the congestion involves the whole brain or part of the brain. The treatment of congestion of the membranes, if it can be recognised, is that of meningitis.

INFLAMMATION (MENINGITIS).

Of the three membranes that enclose the brain, only two are pathologically separable, since the arachnoid and pia mater always suffer together. The separate inflammation of the dura mater is termed "*pachymeningitis*," and is much less common than the affection of the pia arachnoid, which is commonly meant when "meningitis" is spoken of. The affection of the soft membranes has been of late termed "*leptomeningitis*," in more precise antithesis to pachymeningitis.

INFLAMMATION OF THE DURA MATER (PACHYMENINGITIS).

In the dura mater there are two layers; a thin inner layer with a smooth epitheliated surface, and a thicker, looser, outer layer, which serves as a periosteum for the bones. Inflammation may affect primarily either of these layers, and thus constitute what has been termed external or internal pachymeningitis. The former is the most common.

External pachymeningitis is almost always secondary to injury or to adjacent disease, and is very rare in children. The injury that causes it is usually a blow on the skull, often one that fractures the bone, and causes an effusion of blood between the bone and the dura mater. The disease is usually caries or necrosis of the bone, sometimes involving only the outer table; in caries of the petrous bone there is often purulent pachymeningitis in the neighbourhood, which is believed by some to be the agency by which the pia mater becomes inflamed in these cases. The disease results, in rare cases, from mischief outside the skull, as erysipelas, probably by the mechanism of the veins of the diploë. Very rarely the meningitis occurs without any traceable cause. The anatomical changes consist at first in redness and œdematous swelling of the tissue, which soon becomes first discoloured and then infiltrated with pus, which may accumulate in considerable quantity between the dura mater and the bone. Very rarely pus forms between the two layers of the dura mater. The inflammation may spread to the inner layer, and even from this to the pia mater, so that the two become glued together by lymph. If the inflammation subsides without the formation of pus, there remains a thickening of the outer layer of the dura mater, firmly connecting it with the skull, and into this tissue osteophytic growths may extend from the bone.

The *symptoms* of the secondary inflammation are usually lost in those of its cause. When distinct symptoms exist they are usually gradual in their onset, and consist in headache, delirium, and sometimes convulsions. When there is a collection of pus compressing the motor region of the cortex, there may be paralysis of the opposite limbs. There may be fever, but often there is no elevation of temperature that can be referred to the meningeal inflammation. The condition is serious, but not quite so grave as most other forms of meningitis. The *treatment* is first and chiefly that of the local disease or injury exciting the intra-cranial inflammation; and secondly, the same measures as are suitable for other forms of meningitis, in so far as they are compatible with the treatment of the cause of the disease. If there is reason to suspect the formation of pus between the bone and the dura mater, this may be let out by trephining.

Internal pachymeningitis is met with in two forms, purulent and hæmorrhagic, the latter giving rise to what has long been termed "hæmatoma of the dura mater." Both are uncommon; the former, indeed, is extremely rare. In congenital syphilis the whole membrane may be much thickened by fibroid tissue.

Purulent internal pachymeningitis is usually associated with a similar inflammation, either of the outer layer or of the pia mater. In one or two cases it has been met with as an apparently primary condition. The symptoms have been similar to those of purulent inflammation of the pia mater.

"HÆMORRHAGIC INTERNAL PACHYMENINGITIS;" HÆMATOMA OF THE DURA MATER; MENINGEAL BLOOD-TUMOUR.

A peculiar formation that extends over the brain, commonly over both hemispheres, and consists of membranous layers with the remains of blood between them, has long been known by this name. The malady is very rare, and it is, indeed, of interest chiefly as a pathological curiosity enigma, for it has not been often suspected during life, and we do not know whether it can be influenced by treatment. It is commonly described as a result and variety of inflammation of the dura mater, because Virchow, in 1854, brought forward evidence to show that this was its origin. It had previously been ascribed to primary hæmorrhage,* and this view, advocated by Prescott Hewett in 1845,† has been recently revived by Huguenin and strongly supported by Wigglesworth; the question is still undecided. But provisionally, and on account of the course of the symptoms, the disease may be considered among the varieties of inflammation. It is so uncommon‡ that a brief notice of it may suffice; Huguenin's exhaustive description of the malady in Ziemssen's 'Handbuch'§ and Wigglesworth's account of it‖ are accessible to all readers likely to be interested in the subject.

CAUSES.—The condition is met with chiefly in males, less than one fourth of the cases having been in females. It is least rare in old age; more than half the cases occur in persons above fifty years of age, 40 per cent. are over sixty, and just a quarter are over seventy. Considering the comparatively small proportion of persons who reach the age of seventy, this proportion shows that the tendency to the disease is enormously increased by the tissue changes incidental to age. In early life it is relatively as well as absolutely infrequent, and occurs chiefly in infancy, as frequently in the first year of life as between one and twenty years of age. The affection is commonly secondary; some cases have followed an injury to the head, but the diseases to which it is most frequently consecutive are some forms of chronic insanity (especially general paralysis of the insane) and chronic alcoholism. In general paralysis it occurs at any age over thirty. It is said also to have followed some acute and chronic blood diseases, especially acute rheumatism and smallpox, typhus and relapsing fevers, scurvy, and profound anæmia. A similar condition has been met with in "scorbutic rickets," in which periosteal hæmorrhages occur.

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* By Houssard, Baillarger, and others.

† 'Med.-Chir. Trans.,' 1845.

‡ Its rarity, at any rate outside asylums, may be judged from the fact that during the first forty years in which the Pathological Society received the curiosities of metropolitan necroscopy, not a single specimen was brought before the Society from any London hospital.

§ Bd. xi, p. 342 (1st ed.); vol. xii of the American translation.

‖ 'Brain,' 1892. See also Pitt, 'Path. Trans.,' 1892.

PATHOLOGY.—The condition is bilateral in about half the cases. Within the dura mater, between it and the arachnoid, and commonly adherent to both, are layers of peculiar membranous tissue. This extends over the greater part of one or of both hemispheres. The tissue is soft and red at first, afterwards paler and firmer. There may be several layers, even six or seven, adherent at the edges, so as to form a series of sacs, which contain blood, liquid or coagulated, or in various stages of degeneration; and ultimately there may remain only coloured serosity, in which crystals of cholesterine are sometimes found. Occasionally the membranes are adherent in places, so as to render the cavities loculated. The red tint of the membranes depends on minute vessels, and on the presence of degenerating blood; the increased consistency and pallor of the later stage is due to the development of a delicate fibrous tissue. In rare cases, suppuration has taken place so as to convert the whole mass into an abscess, in which, however, the membranes and vessels can be traced to the dura mater, so as to demonstrate the nature of the lesion.* The nature of the earliest stage of hæmatoma is a matter on which some doubt exists; as already indicated, Virchow finds in it a process of inflammation, and believes that a delicate vascular membrane is formed by inflammation, and that into this hæmorrhage takes place. Huguenin found simple hæmorrhage as the initial change in some cases of general paralysis, and it has been advocated by many subsequent investigators; the facts ascertained by Wigglesworth are of especial importance.† Some of the veins of the surface of the brain (arachnoid) may be normally attached, for part of their course, to the dura mater, and leave it to open into the superior longitudinal sinus. They vary in number, but are abundant in the fœtus and newly born child. In some cases many of them persist, and their walls are prone to degenerate, especially if, from any cause, the brain undergoes atrophy, and thus their external support is lessened, while their walls are exposed to an undue amount of strain, before which their nutrition fails. Such failure is also supposed to occur in the acute diseases with a hæmorrhagic tendency in which hæmatoma is met with, such as the "scorbutic rickets" mentioned above. But other observed facts show that an inflammatory membrane may be formed, and thus support Virchow's opinion. (See "Serous Pachymeningitis," p. 328.)

SYMPTOMS.—In some cases of hæmatoma in general paralysis, no symptoms have been present other than those that are common in this disease when no unusual condition is found after death. In other cases there have been apoplectiform seizures, recurring, and ascribed to the hæmorrhages that occur from time to time in the cavities of the hæmatoma, and there have been more persistent symptoms of depressed cerebral function—somnolence or coma, with contracted pupils and general muscular weakness. Nystagmus is not uncommon, and optic neuritis sometimes occurs towards the close. In the cases that follow injury, mental and physical weakness develops, and the patient is brought almost into the condition of the insane subjects of the disease. Headache has been a prominent symptom in many cases, and has been sometimes limited to the vertex or to the temporal regions in the early stage. Vomiting is occasionally frequent throughout the course of the disease. Convulsions are not common, but have occasionally occurred, generally on one side. In many cases there is hemiplegia, seldom complete, with or without early contracture. The unilateral symptoms occur when the disease is confined to, or greater on, one hemisphere of the brain. In children the disease is said to run an acute course, with convulsions and sometimes fever. It is doubtful whether, in them, the malady can be diagnosed, unless chronic cerebral symptoms, convulsions, twitchings, hemiplegic weakness, &c., occur in the subject of hæmorrhagic rickets. In other cases, symptoms of impairment of the functions of the brain and headache, following or coinciding with a cause of hæmatoma, may lead to a suspicion of its existence, and this may be strengthened by the occurrence of apoplectic seizures. But all the symptoms of hæmatoma (at any rate, when it is unilateral), including the apoplectic seizures, may be caused by a glioma of the brain-substance. The malady has caused death in most instances. One case of recovery is on record: the subject was a drinker, who was murdered six months after the cerebral symptoms had passed away, and in whom the remains of the disease were found.

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* *E.g.* Max Klink, Inaug. Diss., Greifswald, 1890.

† 'Journal of Mental Science,' 1889.

Diagnosis.—The symptoms of hæmatoma are not in themselves decisive, although they may raise a suspicion of its existence from their association with a known cause of the condition, especially general paralysis of the insane and chronic alcoholism. An injury may give rise to other lesions, manifested by analogous symptoms, so that it is of comparatively small significance. But with either of the first two conditions, the development of chronic muscular weakness, with mental failure if it did not before exist, rigidity of the limbs, paroxysms of tonic spasm, sometimes with unilateral disturbance, as hemiplegia and conjugate deviation of the eyes, justifies a diagnosis. If a knowledge of the symptoms were more general, it is probable that the malady would be more often suspected than it now is, outside the walls of asylums for the insane.

Prognosis.—If the symptoms are so pronounced as to enable a probable diagnosis to be made, they will almost certainly increase and have a fatal result; only when the existence of the malady is suspected at an early stage, and in an alcoholic patient, is there any chance of its arrest.

Treatment.—The part which extravasation of blood manifestly takes in the production of the morbid state suggests treatment calculated to arrest hæmorrhage—rest, elevation of the head and shoulders, and the application of cold to the head. Moderate purgation and diuresis may also be employed. The removal of the cause is necessarily essential. The possibility that inflammation constitutes one element in the causation of hæmatoma suggests the measures suited to chronic meningitis; but treatment is generally powerless to arrest the course of the disease, and it is doubtful whether depletion does other than harm.

Serous Pachymeningitis is a very rare condition, met with in young children and occasionally in general paralysis, in which a membranous layer lines the dura mater, and is continuous at the base with a thinner layer that covers the arachnoid. The two are separated by fluid, which has led such cases to be sometimes described as "external hydrocephalus." The microscope may reveal a layer of extravasation in the outer membrane, when no hæmorrhage can be seen with the unassisted eye. In children, enlargement of the spleen, from "peri-splenitis," has been found associated. Enlargement of the head, and compression-atrophy of the brain, may or may not be present. As a rule, the mind is not affected, and cerebral symptoms are slight (Henoch); but if the brain is small and indurated, there may be considerable mental deficiency. Irregular periods of pyrexia and redness of the scalp may be present.*

INFLAMMATION OF THE PIA MATER (PIA ARACHNOID); MENINGITIS, LEPTOMENINGITIS.

Meningitis may be acute or chronic, and apart from this fundamental difference, certain varieties are commonly distinguished, according to (1) its seat, whether this is at the convexity, or at the base, or in the ventricles; (2) its origin, whether primary or secondary to a local cause or to a general disease; (3) its nature, whether simple, purulent, tubercular, or syphilitic. The symptoms of these forms present certain differences, but yet have much in common, and in all cases the problem in diagnosis is first to ascertain the existence and seat of meningitis, and then to distinguish its nature and origin. Hence it is more useful to consider all forms together, noting, as we go on, the differences between them, than to give a separate account of each variety. Four fifths of the statements that must be made about each are true of all. Epidemic cerebro-spinal meningitis is separately described, although it is not sharply separated from the simple form.

Causes.—Acute meningitis occurs at all periods of life, but is most common in children under ten. Only one form is less frequent in children than in adults—meningitis of the convexity; and this is due chiefly to its common traumatic origin. Taking all forms together, the disease is more frequent in males than in females, but the several varieties exhibit some differences in this respect. Hereditary tendency is active chiefly in causing the tubercular form. Station in life, occupation, climate, and season are influential only to a small extent as determining some of the causes.

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* Wheaton 'Trans. Path. Soc.,' 1891.

Direct Causes.—(1) *Traumatic influences* of various kinds, acting on the membranes by laceration, hæmorrhage, or concussion.

(2) *Adjacent disease,* especially that which is attended with suppuration, often causes purulent meningitis by direct extension of the pathogenic virus. The adjacent disease may be (*a*) outside the dura mater, as injury and disease of the bone, especially caries of the petrous bone and disease of the ear; or it may be outside the skull, as erysipelas and other suppurative diseases of the scalp. In several recorded cases inflammation of the eye has set up a fatal meningitis, and a still more frequent cause is inflammation of the upper part of the nasal cavities. Between these and the meninges there are free communications, which are described in the chapter on "Abscess of the Brain." Growths in the bone above the nose have been found to afford a ready means for the entrance of organisms. (*b*) The adjacent disease may be in the brain—a tumour or an abscess, very rarely hæmorrhage or softening. An abscess may cause meningitis without rupture, but rupture always causes intense purulent inflammation. A superficial growth commonly causes some meningitis, of which the acute symptoms are frequently perplexing.

(3) *Acute disconnected diseases* are sometimes attended with meningitis. (*a*) Acute specific diseases: measles, scarlet fever, smallpox, typhoid fever, acute rheumatism. The complication is, however, rare in any of them, its existence being often erroneously assumed from mere intensity of delirium. It is least rare in smallpox, perhaps on account of the septicæmic influence of the extensive cutaneous suppuration. It is rare in typhoid fever, according to pathological evidence; but it does sometimes occur in simple form, especially over the convexity. I have also known tubercular meningitis to develop during the fever. (*b*) Meningitis has a special relation to acute pneumonia, and is probably due to the cause of the inflammation of the lung, especially to the specific "diplococcus" which has commonly been found in the inflamed lung since Fraenkel discovered it. The same organisms are found in the meningeal exudation. This inflammation is usually purulent, and the organisms connect it, as we shall see, with the epidemic cerebro-spinal form. (*c*) Septicæmia often causes meningitis, whatever be the source of the blood-poisoning, whether external injury, internal abscess, softening of caseated glands, septic puerperal processes, the softening of clots, or ulcerative endocarditis. The effect is probably produced through the agency of an organised virus, and a minor factor in the process may be the micrococcal embolism of minute vessels; it is not uncommon in such cases to see minute extravasations in the pia mater, with pale centres, such as are seen in the retina in

similar cases. Sometimes local meningitis exists near the plug in a large vessel, when the source of the plug is a septic form of endocarditis. It probably follows other toxic influences received from without. Thus I have known fatal meningitis to follow exposure to "bad smells" on a search for their source. From all these septic causes the inflammation is usually purulent.

(4) Insolation is an occasional cause, and influences chiefly the convexity. The meningitis that results is usually simple, and not purulent. In cases of rapid death, only indications of congestion may be discoverable.

(5) Excessive mental work and mental excitement are usually regarded as occasional, although rare causes. A predisposition is probably necessary to make this influence operative, and the effect of mental work may easily be over-estimated.

(6) *Specific Processes.*—The most common cause of acute meningitis is the growth of tubercles in the membranes, which is usually attended with inflammation, the intensity of which varies much in different cases. Syphilis is also a cause of meningitis; although the inflammation is usually chronic, a very acute form, yielding to treatment, is occasionally met with.

Lastly, meningitis may occur, either at the convexity or the base, for which no cause can be discovered beyond some impairment of the general health. It is possible that ill-health may permit organisms, which would otherwise be destroyed, to persist and assume a virulent development.

Causation in Relation to Place.—Meningitis limited to the *convexity* may result from local adjacent disease, from insolation, or from general disease (acute specifics, pneumonia, and septicæmia); but in the last class the inflammation is often general, and involves the membranes at the base as well as those over the convexity of the hemispheres, and frequently also the membranes of the spinal cord. Meningitis limited to the *base* is rare except in association with tubercles, basal growths, disease of bone, or adjacent disease, but occasionally results from syphilis. Simple basal inflammation may be precisely like that caused by tubercles. Limited *ventricular* meningitis is met with occasionally in young children, chiefly during or after acute diseases, such as the exanthemata; in adults it is extremely rare.

Causation according to Nature.—Simple meningitis may result from any of these causes, except perhaps septicæmia; the etiological facts just stated are therefore, in general, applicable to this form.

Purulent meningitis, in which pus is formed from the first, is usually the result of adjacent suppuration or of septicæmia. The causes of the former have been already enumerated (see also "Abscess"). A septic thrombus in a vein or sinus is sometimes the mechanism by which such meningitis is excited.

Tubercular meningitis occurs at all ages, but is most common in children, especially between the ages of two and ten years. In infancy it is not common, although it has been met with as early as six weeks after birth. It is not rare in early adult life, and is occasionally met with in middle life, but scarcely ever during the later period. In children, general tuberculosis almost always exists, but the indications of this may be slight, and the meningeal disease may have the aspect of a primary affection. In adult life, distinct indications of phthisis usually precede the meningitis, but are sometimes absent in youth, in whom, however, some scrofulous or caseating process commonly exists, and is a focus of blood-infection. It is probable that there is little difference in the proclivity of the two sexes, at any rate after puberty. It is generally thought that in childhood boys suffer rather more frequently than girls.* Hereditary tendency is of unquestionable importance. Most cases occur in families in which there is a clear history of phthisis or of infantile tuberculosis, sometimes evidenced chiefly by the early death of many children. The disease is common in all classes of society. Imperfect nourishment and bad air no doubt aid in its development, but the cases are very numerous in which no accessory cause can be traced. A distinct excitant is also rare, although a blow or fall on the head seems distinctly influential. Brain-work may assist, but only in the subjects of the general disease. Any depressing physical influence, including maladies of different nature, may have the same effect; thus it occasionally occurs as a sequel to some other acute disease, especially measles (the influence of which in exciting tuberculosis is well known), whooping-cough, broncho-pneumonia, diarrhœa, typhoid fever, &c. A blow or fall which would otherwise cause trifling and transient inflammation may excite fatal tubercular meningitis in those who have even caseous deposits in some locality. Very seldom it comes on in the midst of apparent health; more often the child has been distinctly "ailing" for a month or two before the onset.

Chronic meningitis may be the sequel of an acute attack. Apart from this, diffuse chronic meningitis is generally the result of alcoholism; occasionally it is due to injury. Focal chronic meningitis, when not traumatic, is syphilitic in most cases; possibly, in later life, it is sometimes due to gout. In children, chronic basal meningitis is not uncommon in the posterior fossa.

ANATOMICAL CHARACTERS.—The changes may be found in all parts of the membranes, or only at the convexity or the base. When the cause is adjacent disease, the neighbouring region of the membranes may alone suffer. Certain changes are common in all forms of meningitis. The earliest is a diffuse reddening of the pia mater, due to the injection of finer vessels than are distended by the mechanical influence of the mode of death. Soon there occurs opacity of the membranes, recognisable most readily in the arachnoid. It occurs also along the vessels of both arachnoid and pia mater, distinctly in consequence of distension of their lymphatic sheaths. It is well seen over the sulci of the convexity, and the spaces at the base of the brain. Collections of yellowish-white or semi-purulent lymph form at these parts and around the nerve-trunks, the sheaths of which may be distinctly reddened. In the early stage of purulent meningitis these collections may be punctiform, and may resemble very closely tubercular granulations in the pia mater; by coalescence they constitute irregular semi-purulent areas. In cases of some duration the purulent spots may have undergone caseation or induration, the latter chiefly in simple meningitis. The nerve-trunks are often involved, and under the microscope the inflammation may be found either limited to the sheath or infiltrating the substance of a nerve that has been paralysed. Minute hæmorrhages may be seen in the nerve, and its fibres may be in various stages of acute degeneration. Sometimes no change can be found. The inflammation may spread on the one hand to the dura mater, and on the other to the substance of the brain. The inner surface of the dura mater may be reddened, and even covered with a layer of lymph, which may glue it to the pia arachnoid. Its outer surface is often the seat of purulent inflammation in cases due to adjacent bone disease. The superficial layer of the brain is often reddened, and even spots of softening may be seen within it. Sometimes it contains punctiform hæmorrhages. The fluid in the subarachnoid space is increased in quantity, and is often turbid. Indications of inflammation may also be present in the ventricles; the lining membrane may be swollen and opaque, sometimes even covered with a layer of lymph (*ependymitis*); the subjacent brain tissue may be softened. The choroid plexus and velum interpositum may also present signs of inflammation. The lateral ventricles often contain a great excess of fluid, which may be turbid, with flocculi of lymph. It may be so considerable as to distend the ventricles and flatten the convolutions, a circumstance that suggested the name "acute hydrocephalus" formerly applied to all these cases. The ventricular effusion seems to be the result, in some cases, solely of the ventricular inflammation, but often there is also a collection of lymph about the openings by which the fourth ventricle communicates with the space around the brain, and the obstruction to these must increase or cause the ventricular distension. In the so-called ventricular meningitis, effusion into the ventricles and slight traces of inflammation are all that is found; the external membranes are normal. Although the traces of inflammation are slight, the acute and febrile course of the disease is consistent only with its inflammatory nature. In all forms, if the inflammation of the ependyma passes away, permanent thickening of the lining membrane may remain, and there may even be adhesions closing the cavity of the posterior cornu. A permanent excess of liquid may be left in the cavities.

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* The Registrar-General's returns for the twenty-five years ending 1872 give the deaths from hydrocephalus in males under five as 91,681, and females 66,708. Probably the chief cause of death returned as "hydrocephalus" at this age was tubercular meningitis.

In purulent meningitis the membranes are covered with a layer of greenish-yellow, often fœtid pus, sometimes at the convexity, less commonly at the base only, often at both. The purulent inflammation frequently extends down the membranes of the spinal cord, sometimes as low as the cauda equina, and in one recorded case suppurative inflammation in the orbit was secondary to that within the skull.* The surface of the brain is often softened, and small collections of pus may be found within its substance, apparently secondary to the meningeal suppuration. Very rarely primary purulent inflammation has been limited to the ventricles.

In *tubercular meningitis* the inflammation is never actually purulent. The appearances are usually those of the simple inflammation already described, conjoined with the characteristic tubercles. The amount of jelly-like and semi-opaque lymph is often considerable, but it is found chiefly at the base, and is generally most abundant about the optic chiasma, between this and the pons, and in the commencement of the fissures of Sylvius. The membrane over the convexity has a dry aspect, but to the finger may feel somewhat sticky; and if a scalpel is passed over the surface, it removes a little pyo-lymph. The two opposed surfaces of the hemisphere, in the longitudinal fissure, may be slightly adherent where in contact. There is often an excess of arachnoid fluid at the base of the brain, and then the sheaths of the optic nerves are usually distended, so as to form a pyriform swelling behind each eye. Ventricular effusion is present in the majority of cases (about four out of five); generally the quantity does not exceed a few ounces, but it may be so large as to compress the cortex, and to distend all the ventricles. The indications of closure of the communication between the fourth ventricle and the surface, already mentioned, are more frequent in tubercular than in simple meningitis. The lining membrane of the ventricles and the choroid plexuses may be distinctly inflamed. In addition to the signs of inflammation in the membranes, these present the distinctive characteristic of this variety, miliary tubercle. The granulations are most easily seen where the inflammation is slight or absent, especially on the under surface of the temporal lobe. They are at first very minute, and so transparent that they can often be detected only when their prominence is recognised by looking obliquely at the surface. Although seated in the pia mater, they cause a prominence of the arachnoid where this covers the pia mater closely, resembling the minute air-bubbles beneath the arachnoid that are often produced during the process of removing the brain; these disappear if the finger is passed over the surface, while the tubercles remain unchanged. In other places they are of larger size and semi-opaque, and in some places several are aggregated together. Where the lymph is abundant, only the larger and older tubercles can be recognised. The microscope shows the granulations to consist of lymphoid cells, situated usually around a vessel, within its perivascular sheath. It is important to remember that meningeal tubercle and tubercular meningitis are not quite identical. Tubercles may be found in the membranes when there is no sign of inflammation, in cases of general tuberculosis, and they may be accompanied by symptoms of cerebral disturbance resembling those caused by inflammation. When inflammation accompanies them, its extent and degree vary. It is usually considerable only at the base, and is scarcely ever seen at the convexity and not at the base; not rarely it is general. Sometimes the inflammation is confined to one small area, as, for instance, to the neighbourhood of the central convolutions on one side; such local meningitis is usually associated with tubercles of some size: very rarely inflammation and tubercles of the ordinary character are confined to the convexity of one hemisphere.* Even when the inflammation is apparently confined to the base, tubercles can usually be detected, often in abundance, over the convexity, but they are still more numerous, larger, older, and more opaque at the base, especially about the commencement of the Sylvian fissure.

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* Eröss, 'Cent. f. Nerv.,' 1883, 221.

The characteristic bacilli of tubercle have been found in the pia mater in these cases (Cornil and others). Moreover the organisms have been found when no tubercles could be seen with the naked eye, although commencing granulations were discovered by the microscope,† and even in the grey substance of the cortex beneath a focus of limited inflammation.‡ Tubercles may sometimes be recognised in the lining membrane of the lateral ventricles, and in the choroid plexuses. A careful examination of the inner surface of the dura mater often shows minute tubercles scattered over it. They are frequently met with in the spinal membranes, dura mater, and pia mater, sometimes one, sometimes both, and occasionally in great abundance, especially over the cauda equina, where the membrane may look as if it had been exposed to a shower of fine sand.§ Signs of inflammation are always slighter in the spinal than in the cerebral membranes, and are often absent, especially when the granulations are upon the dura mater only. The cortex of the brain may be injected and softened at the surface, but if there is much effusion in the ventricles the amount of blood in the vessels of the cerebral substance may be less than normal. Under the microscope the walls of the cortical vessels are often found to be crowded with leucocytes, and sometimes minute aggregations of tubercles may be detected within the cortical tissue. Small extravasations are common, in adults, in the membranes or in various parts of the brain-substance, and are probably due to changes in the walls of the vessels and thrombosis within them.

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* Huguenin, Hilton Fagge.

† Dawson and Hebb, 'Lancet,' 1884, Ap. 12, p. 660.

‡ Déjerine, 'Revue de Méd.,' March, 1885, p. 174.

§ Wortmann found no tubercles in the spinal membranes only in four of twenty-seven cases examined ('Jahrb. f. Kinderheilk.,' Bd. xx, 1883, p. 300).

In these cases a tubercular tumour is occasionally found in the brain, sometimes more than one. I have seen a mass of tubercle the size of a filbert growing from the under surface of the dura mater. Extensive miliary tuberculosis of the small vessels and the substance of the brain, causing almost universal softening, has once been observed.*

In young children tubercles are almost invariably found widely scattered through the other organs of the body. They are most constant in the spleen,—very frequent, also, in the enlarged mesenteric glands. Exceptions to the general character of the affection are rare, but Gee mentions a case in a girl, aged four, in whom a cheesy mass in each lung was the only indication of tubercle outside the membranes, and similar cases have been recorded by others. The caseating scrofulous material, in such cases, was probably the source of the blood-infection, and thus the cause of the meningitis. Tubercles are often to be seen in the choroid in tubercular meningitis, although generally only late in the course, and after its nature has become clear.

Thrombosis in a vein of the convexity is occasionally found in tubercular meningitis, sometimes with the intense secondary congestion of the corresponding region of the brain. It occasionally precedes the formation of visible tubercle. Very rarely a clot extends to a sinus, but the special symptoms of this are usually lost in those of the meningitis.

Chronic Meningitis.—The diffuse, chronic meningitis of alcoholism affects chiefly the convexity, and is always slight in degree. The pia arachnoid is somewhat opaque, especially over the sulci, and there may be lines of opaque lymph along the sides of the vessels. The focal chronic meningitis of syphilis is often associated with a syphilitic growth, or with disease of the walls of the arteries. There is much thickening of the pia arachnoid, with the peculiar exudation—at first "gummy" in aspect, while, later, caseous foci are scattered through it—which glues all adjacent structures together, and often unites the pia mater to the dura mater. It may be so abundant at a certain spot as to occasion doubt whether it should not be regarded as a "gumma," with secondary inflammation. Ultimately, if caseation is prevented by treatment, fibroid transformation may occur, and a thick layer of tissue, tendinous or cartilaginous in aspect, extends over a certain region, more often at the base than at the convexity, surrounding and compressing the nerves, and uniting the various membranes. The thickness of the layer may be as much as a third of an inch; the dura mater is often also thickened.

In children, chronic inflammation occurs chiefly in the posterior fossa, about the pons, medulla, and cerebellum, which are often firmly adherent. In another chronic form, which is perhaps only a later stage of the syphilitic inflammation, the inflammatory tissue has undergone fibrous transformation. When chronic inflammation involves the membranes about the medulla oblongata, it generally closes the openings of the fourth ventricle, and internal hydrocephalus results.

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* Gee, 'Reynolds' System of Medicine,' vol. ii, 2nd ed., p. 408.

Symptoms.—Certain symptoms occur in meningitis irrespective of its seat or form, while others depend on its situation, whether at the base or over the convexity of the brain; the special pathological forms differ in their course, and in the relative prominence of various symptoms. In every form and position, the manifestations of the disease present wide diversity in different cases; yet, as a rule, certain symptoms are present, and develop in such a manner as to be fairly characteristic.

The symptoms consist partly in general disturbance, such as attends any local inflammation, partly in cerebral symptoms, some general and others local. In many cases there are also associated symptoms due to the pathological process to which the meningitis is secondary. The chief symptoms common to all forms will be first described.

Premonitory symptoms are often present, although never distinctive in character. There may be such general indisposition as may precede any malady, languor and malaise, to which is sometimes added mental irritability. In children, vomiting without cause, or on some slight indiscretion in diet, often precedes other symptoms for some days. Occasionally prodromata are absent, and the pronounced symptoms of the disease set in suddenly.

The most important of these are headache, delirium, vomiting, convulsions, rigidity and weakness of the limbs, and paralysis of cranial nerves, strabismus and inequality of the pupils, coming on acutely or subacutely, but not suddenly. They are usually accompanied by elevation of temperature, and followed in a few or many days by somnolence and coma.

Headache is, of all symptoms, the most common, and is usually one of the earliest; it is prominent throughout, until the patient becomes unconscious. It is most frequently frontal, but sometimes general. Continuous in some degree, severe exacerbations occur, in which the pain is very intense, and may cause shrieks of suffering, which have received the name of the "hydrocephalic cry;" and these may continue even when the patient is almost unconscious. In rare cases, meningitis runs its course with little or no pain, especially the secondary purulent meningitis of septicæmia and in the simple meningitis of some other blood-states. Vertigo is occasionally complained of, especially at the onset, but on the whole is not frequent.

Delirium is another important symptom. It may be either slight and quiet, or active and violent. It is often an early symptom, although it chiefly occurs at the onset of the disease, and only in those who are specially predisposed, and then may have its character modified by the predisposition, such as alcoholism or hysteria. Diagnostic errors are then easy. The delirium, as a rule, derives its significance from its association with headache. At first there is merely wandering during sleep, and, as the mental disturbance increases, periods of perfect or imperfect consciousness alternate with periods of delirium, when the cries of pain are often frequent and severe. When it has set in, it usually persists, sometimes with intermissions, until the coma comes on.

Vomiting consists in the rejection of the contents of the stomach, often without nausea; there is not usually retching when the stomach is empty. It is a frequent, and often a very early symptom, whatever be the seat of the inflammation. The tongue is at first clean, but it may afterwards become thickly furred and the breath very offensive. The bowels are confined, often obstinately, and the abdomen is retracted.

General convulsions occur, irrespective of the seat of the inflammation, but are rather more frequent in children than in adults. They may occur at any time during the course of the disease, from the onset to the end. They are occasionally almost the only symptom of the meningitis that is produced by septic processes. Rigidity of the muscles of the neck, with retraction of the head, is common in meningitis about the posterior part of the base, and may extend to the muscles of the back if the inflammation passes down the spinal canal. It is a symptom of very great importance, being frequently the first unequivocal indication of the malady.

Optic neuritis is a common symptom of meningitis of the base, but is rare when the inflammation is confined to the convexity. It is not an early symptom, being seldom met with until the end of the first week. I have once seen it (unilateral) as early as the fifth day after the onset of the symptoms of tubercular meningitis. It is due to the extension of inflammation from the membranes to the sheath and substance of the optic nerve within the skull, and the descent of this inflammation to the eye, probably aided by the passage of material down the sheath of the nerve to the neighbourhood of the globe. The intra-ocular changes are seldom intense. There is swelling of the papilla, blurring the edges of the disc, and often (especially in tubercular meningitis) the swelling is paler than in commencing inflammation from other causes. The veins are full, but hæmorrhages are rare, and the neuritis is never so intense as in cases of tumour. Choroidal tubercles are sometimes seen in the tubercular variety.

Hyperæsthesia of the skin is occasionally met with, and that of the special senses is very common, so that light and noise distress the patient and increase his suffering. Both are apparently due to the general excitability of the brain. Retention of urine is common in the later stage, when consciousness is obscured. Occasionally there is incontinence of urine and fæces.

Local symptoms occur in the cranial nerves and limbs. In the former they depend chiefly on the affection of the base, in the latter on that of the convexity. These symptoms are partly those of irritation, partly those of paralysis. The most common symptoms are in the ocular nerves, causing alteration in the position or movement of the eyeballs or in the size of the pupils. Strabismus is a very important symptom; it is often at first transient, or present only on movement, and even then inconstant, depending apparently on varying overaction of a muscle. In other cases there is persistent and evidently paralytic weakness of certain muscles. Slight ptosis may occur, but complete paralysis of the third nerve is not common. Indeed, the whole of any nerve is scarcely ever paralysed unless other nerves suffer in some degree—a point of some importance. The pupils are usually contracted in the early stage, especially when there is intolerance of light; subsequently they are often dilated. Inequality is a very common and most important symptom; it occurs in inflammation of the convexity as well as of the base. It is often transient and variable, present at one time and absent a few hours later; now one pupil, now the other, may be the larger. An undue sensitiveness of the dilator fibres to cutaneous stimulation has been noted by Parrot.

Next in frequency is an affection of the facial nerve, weakness of which is indicated by inequality of the mouth, or distinct defect of movement. When the disease is at the base, all parts of the facial nerve may be affected; when it is over the convexity, the lower part suffers alone in most cases; perhaps there is occasionally brief weakening of the upper part. The muscles of mastication may be rigid, but this is not frequent, and their paralysis is still more rare. The hypoglossal nerve sometimes suffers, and deviation of the tongue results.

The hyperæsthesia of the special senses has been already mentioned. An affection of the olfactory nerve is very rare. So also is considerable impairment of sight, which scarcely ever occurs except in cases in which the inflammation passes into a chronic stage, and damages the optic nerve considerably, partly directly, and partly by the contraction of lymph that forms around it. The auditory nerve suffers more frequently, usually in association with the facial (which lies beside it at the base of the brain), but in greater degree. Both auditory nerves are sometimes supposed to be damaged alone in meningitis associated with bilateral otitis interna, but it is certain that in these cases the deafness is due to the labyrinthine inflammation, and not to the meningitis.

One-sided symptoms are not uncommon—muscular rigidity, unilateral convulsion, or hemiplegia. Complete hemiplegia is rare, and occurs only when the membranes about the central convolutions are involved. Sometimes no difference can be seen in the state of the membranes over the two hemispheres to account for the difference in the limbs; the paralysis is apparently due to arrest of function by the irritation. In rare cases the hemiplegia is due to the occurrence of thrombosis in a surface vein. Rigidity of the limbs of one side or of both is very frequent. It may be slight or considerable, and often varies from time to time; passive movements then cause pain, and the pain produced is often greater than the amount of rigidity would suggest. Convulsions, beginning locally, or partial in extent, are also common in meningitis of the convexity. Unilateral hyperæsthesia may also occur, and there may be areas of anæsthesia, but complete hemianæsthesia is very rare. Aphasia occurs chiefly in the tubercular variety, perhaps because such inflammation is apt to extend along the fissure of Sylvius. It is occasionally an early symptom.

The pyrexia of meningitis varies much in different cases. Often there is from the first a considerable elevation of temperature, 101°—103°, and it remains high, with irregular variation, throughout the disease. In the most acute purulent form the temperature may rise quickly to 104° or 105°, and remain high till death. In other cases the temperature is at first raised and towards the end falls below the normal, and a rectal temperature of 96° or 97° has been observed. On the other hand, towards the end the temperature may be very high, 106° or 108°. It is said that, in very rare cases, the disease runs its course without fever, and it is certain that for some days the temperature may not be above the normal; apparently this is the result of an influence exerted by the nervous system which neutralises the tendency of the inflammatory process.

The pulse presents as wide variations as the temperature, but there may be no correspondence between the variations in the two symptoms. In some cases it is frequent throughout; more often it is slow, 68, 50, or even 40, and it is often not only slow but irregular in rhythm. The pre-mortem rise in temperature is usually attended by extreme frequency of pulse, 160, 180, and sometimes uncountable rapidity.

The vaso-motor system is often disturbed. The well-known *tache cérébrale*, in which cutaneous irritation is followed by unusually vivid and enduring congestion of the skin, has been erroneously supposed to be of special diagnostic significance, but it is met with in many other affections. There is often a strong tendency to the formation of sloughs and bedsores, and these vesications are frequently produced by the application to the skin of hot bottles, the heat of which is insufficient to cause the effect in a healthy person; uncovered metallic bottles are especially dangerous. These trophic changes are more frequent in subacute than in acute meningitis.

Respiration is sometimes slightly quickened, but it is rarely much disturbed until the final period, when in tubercular meningitis the Cheyne-Stokes rhythm is often observed, and, in this disease, is always of fatal significance. More frequent are irregular pauses, in which the child ceases to breathe for several seconds. In meningitis of the posterior fossa there is sometimes almost sudden failure of breathing, irregular feeble respiration, with rapid cyanosis, quickly followed by death.

The urine is scanty and febrile in character. Albumen or a trace of sugar sometimes appears.

Course.—The onset is sometimes rapid and violent, marked by sudden and high pyrexia, by rigors, and by special symptoms of intense degree. In such cases the inflammation is usually purulent; the spinal membranes are commonly involved, and the cases resemble the epidemic form. The course of these cases is sometimes so rapid as to resemble that of the slower forms of cerebral hæmorrhage. Death may occur within forty-eight hours after the onset of the symptoms. In other cases the onset is gradual and insidious, and this is especially frequent in tubercular meningitis. During the early period, symptoms of excitement of function predominate; delirium accompanies the headache, there are convulsions and muscular contractures. At a later period there is depression of function, somnolence deepens to coma, and various paralyses occur. These are often distinguished as first and second stages of the disease, and the deep coma that precedes death is regarded as a third stage. These stages are not always distinctly recognisable. Coma sometimes develops almost at the outset, and palsy of limb or face may be the earliest symptom. Often the local symptoms are trifling, and only the general cerebral symptoms are marked.

The duration of meningitis varies from two or three days to as many weeks. The most rapid cases are those due to septic processes, and cases of the primary purulent inflammation already mentioned. But the maximum duration mentioned above is occasionally exceeded, and cases are met with that must be regarded as subacute. They are generally examples of simple inflammation, and occur chiefly in children; they constitute a transition to the form of chronic infantile meningitis mentioned on p. 346.

Symptoms in Relation to Locality.—In meningitis of the convexity delirium is more pronounced, local convulsions and hemiplegic weakness are more common, vomiting is less frequent, palsies of the cranial nerves are for the most part absent, and optic neuritis is rare. In meningitis of the base the cranial nerves suffer early, delirium occurs later, vomiting is frequent, and optic neuritis usually develops before the symptoms have reached a high degree of intensity. When delirium is the chief symptom, the aspect of the case may closely resemble delirium tremens. When meningitis is limited to one part of the base, the cranial nerves suffer, that have their course in that part. There is usually retraction of the head when the inflammation affects the membranes about the pons and medulla—a symptom of very great diagnostic importance. Hemiplegia and convulsions may occur during its course, from the secondary thrombosis already mentioned. Unilateral convulsion, beginning locally, may succeed recovery.

In *ventricular meningitis*, in which abundant effusion and traces of inflammation in the choroid plexus and ependyma are the only morbid changes, the general symptoms are, strange to say, the same as in other forms,—headache, vomiting, fever, convulsions, rigidity, delirium, irregularity of pulse and breathing, and final coma, but the functions of the cranial nerves are seldom interfered with. The fontanelle, if open, is distended and pulsates. Death may occur in a few days, or at the end of ten days or a fortnight. Occasionally, after an acute onset, the symptoms lessen, but the remission is followed by their renewal in fatal intensity. It is said that incomplete recovery may occur, with enduring defect in mind or muscular power, contractures, oscillating gait or convulsions. Enlargement of the head may develop, and the case practically becomes one of chronic hydrocephalus. There is nothing in these symptoms that is distinctive. All are common to meningitis that is external as well as internal, and there is considerable room for doubt as to the accuracy of the diagnosis of cases that are not fatal.

Symptoms in Relation to Form.—In simple meningitis the symptoms are those above described, and their precise character depends on the seat of the inflammation. The course of the disease is often longer than in the other forms—three, four, or six weeks; and the symptoms present greater variations in degree and in character. The rise of temperature is often at first great, but it may subside as the disease passes into a subacute stage. Probably in consequence of the longer course, optic neuritis is frequent and considerable in degree, especially if the base is involved. In meningitis secondary to local disease the seat of the inflammation, and therefore the attendant symptoms, differ according to the position of the disease that excites it. Moreover, the symptoms of the primary malady often mask those of the meningitis which it excites.

Tubercular Meningitis.—The symptoms differ to some extent in children and in adults.

In the child prodromata are frequent. Loss of flesh and indications of general failure of strength are the most common. They are usually due to the process of tuberculosis, of which the meningitis is one result. Slight evening pyrexia often accompanies these symptoms. Other premonitory symptoms are met with in the nervous system,—mental irritability, restlessness at night, and a tendency to frontal headache, the pain being excited by mental work and fatigue.

These symptoms may exist for a few weeks, sometimes for a month or two before the actual onset. When the meningitis is secondary to considerable tubercular mischief elsewhere, as in the lungs, the symptoms of this usually obscure any premonitory symptoms of the meningitis. Occasionally, as the cerebral inflammation develops, the symptoms of the lung affection lessen in a remarkable degree. The headache, which may exist for two or three weeks before the onset of the acute symptoms, is perhaps due to the formation of tubercle in the membranes, if, as is generally believed, this precedes the inflammation. Another antecedent symptom is the vomiting, which may be apparently causeless, or may follow an inadequate cause, *i. e.* it occurs after food that is not quite judicious, although not so indigestible as to be alone a sufficient cause of its rejection. This symptom should always excite suspicion if it occurs without other indications of gastric disturbance, and especially if it is repeated and is associated with other premonitory symptoms.

Such vomiting, more severe in degree, is a common symptom of the onset, which is usually attended by a great increase in the headache, or by the development of headache if this was absent before, sometimes by drowsiness, and often by attacks of general convulsion. In the early stage of the disease the prominent symptoms are usually those of general cerebral disturbance, headache, somnolence, wandering at night, sometimes vertigo, vomiting, constipation, and the conditions of pulse and temperature already described. The rigidity of the neck and retraction of the head are frequent. Aphasia is sometimes an early symptom. At the end of the first week, sometimes earlier, sometimes later, the symptoms in the cranial nerves, already mentioned, come on. About this time also changes in the optic disc are often recognisable. In the course of the second week the somnolence deepens to coma, but in older children this may be preceded by definite delirium, occasionally violent. The other symptoms become more conspicuous or develop. Rigidity of the limbs, local convulsion, or hemiplegia may be added. Defect of sensation is sometimes associated with the palsy, and may be more extensive; it may be preceded by hyperæsthesia. The paralysis may be transient or permanent. The pulse often becomes frequent, 140—180, sometimes suddenly, sometimes gradually. Respiration is often sighing or irregular. Towards the end of this, or in the beginning of the third week, the child lies unconscious and motionless, often with rigidity or flaccidity of the muscles of one side; muco-pus accumulates on the cornea; convulsions are more frequent; the respiration is more irregular, and may present an occasional pause or a distinct Cheyne-Stokes rhythm. The patient may die during the coma with accumulation of mucus in the chest; bedsores may form, and hasten the fatal issue; or death may immediately succeed an attack of convulsions. Sometimes there is an apparent improvement shortly before death, but, as Oxley* has pointed out, the pulse seldom shares in this. The temperature towards the end may sometimes remain at about the same degree of moderate elevation, sometimes it becomes very high, sometimes falls below the normal, and is sometimes very low. Thus in one recorded case on the seventeenth day of the disease, the day before death, the temperature was only 93° (Bokai).

In the cases in which tubercular meningitis affects only the convexity (which are less rare than is commonly supposed) the symptoms in the cranial nerves are absent, vomiting is less obtrusive, and headache and delirium, with convulsions and rigidity of limb, constitute the chief symptoms. Inequality of pupil is often present when other ocular symptoms (including neuritis) are absent, but slight strabismus or diplopia will often be discovered by a careful search, which should include the upward movement. Partial tubercular meningitis, affecting only a small part of the convexity, causes local symptoms resembling those of a tubercular growth, with which, indeed, it is commonly associated.

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* 'Liverpool Med.-Chir. Journal,' July, 1885.

The duration from the onset varies from one to three weeks. It is rarely less than a week, and occasionally as much as four weeks. The average is two weeks. In cases that begin insidiously the duration is determined with difficulty, and if the premonitory period is included the average is more than two weeks. The average duration of tubercular meningitis limited to the convexity is said to be less than when the base is affected. The usual termination is death, but it is not open to question that cases do sometimes recover. Unquestionably cases recover in which the existence of meningitis admits of no doubt, and in which there is a strong presumption that the inflammation is tubercular, although the recovery removes the possibility of rigid proof. In most cases of children who recover from an illness resembling tubercular meningitis, the cerebral symptoms were general pain, delirium, and convulsions; as a rule there have not been symptoms in the cranial nerves beyond inequality of pupil and slight optic neuritis.

In the adult the symptoms of tubercular meningitis are essentially the same as in the child, but, if possible, even more varied in their character and combination. Headache, vomiting, and cranial nerve symptoms are the same. Palsy of ocular nerves is common, but seldom involves the whole of one third nerve (one case in twenty—Seitz); ptosis is more frequent (one in seven). General convulsions are not common, delirium usually occurs earlier, and its association with persistent headache is very conspicuous. The insidious character of the onset is as common as in childhood, in spite of the ability of the sufferer to describe his symptoms. In young women the early stage of the disease is sometimes attended by symptoms of hysteroid character; the morbid functional tendency incidental to the age and sex determines the form of the general functional disturbance that is at first produced by the disease of the membranes.

The disease commonly comes on in the course of phthisis, but the indications of lung disease may be so slight as to escape notice, and sometimes the malady has the aspect of a primary affection, although tubercles are found elsewhere after death. Besides the symptoms already described, it should be noted that hemiplegia, coming on in the course of a few days or a week, and often attended by convulsions, may be the chief symptom when the inflammation involves the convexity of one hemisphere. The duration of the disease in the adult is sometimes the same as in the child, but often it is apparently shorter, perhaps because the early symptoms are overlooked in the presence of the more obtrusive pulmonary disease. Occasionally, on the other hand, it is much longer. Headache generally precedes other symptoms, and the sufferers have often been liable to headache, and increasingly so during the ill-health that preceded the meningitis in most cases. The habitual headache passes into that of the intracranial disease, and increases the difficulty of fixing the commencement of the latter. The fatality of the disease is great, but cases of recovery from what is apparently tubercular meningitis are observed in young adults more frequently than in children. Doubt always hangs over the exact nature of cases that recover, but the evidence of the probable tubercular character of some of these cases is very strong.*

Partial tubercular meningitis is rare. The development of tubercle in the meninges may be local, affecting one part of the pia mater only, and giving rise only to symptoms of irritation of the underlying cortex. When the disease is over or near the motor region of the cortex it may cause convulsion of local commencement or range, and the symptoms are distinguishable from those of a tumour only by their course.

Purulent Meningitis.—The symptoms depend upon its locality. The course is usually very acute, and the pyrexia considerable and constant, often with an evening rise; the temperature is far more regular in its variations than in tubercular meningitis. The general description given of the symptoms of meningitis is in the main

* As an instance of this class may be mentioned the case of a girl aged nineteen, who had lost a brother from caries of the spine, and a cousin from phthisis. For some months she had been growing anæmic and languid, especially after the death of a sister two months previously. Three weeks before I saw her, having previously complained for a week or two of slight headache, this suddenly became intense, chiefly on the left side of the head, keeping her awake at night, making her scream with the pain; it was accompanied by frequent vomiting. After a few days these symptoms lessened, but recurred from time to time, and with especial severity two weeks after the onset, a day or two before I saw her. After this attack she was somnolent, did not speak, and the urine was passed into the bed. The temperature, at first normal, rose to 100°. She moved the right arm less than the left, and had several convulsions with tonic spasm, flexion of the arms, and deviation of the head to the right. When seen, she could be roused to take notice, but not to speak. There was slight rigidity of the limbs, greater on the right side. The legs and upper arms were moved, but not the hands. The pulse was 88 and irregular. There was slight but distinct optic neuritis, in aspect exactly that seen in tubercular meningitis. Her head was shaved, ice applied to it, and a blister to the back of the neck, and phosphate of iron, iodide of potassium, and nitric ether given internally. That evening she had four more convulsions, commencing in the right leg. The day after that on which the blister rose she spoke, and afterwards slowly improved; for several days more, however, urine was passed into the bed, and the right arm was distinctly weaker than the left. She ultimately recovered perfectly.

applicable to the purulent form. But no form of inflammation, not even the tubercular, presents greater variations in symptoms and course in proportion to the intensity of the process. Some cases, secondary to suppuration elsewhere, present extraordinary latency. I have known, for instance, slight occasional strabismus, slight retraction of the head, moderate headache, irregular fever, and optic neuritis to be the only symptoms, although after death both cerebral and spinal membranes were bathed in pus, and the meningitis certainly commenced a fortnight before death. On the other hand, the meningitis that follows rupture of a cerebral abscess causes symptoms of extreme intensity and has a rapid course.

Focal meningitis, involving only a small area of the membranes on one side of either the base or the convexity, is generally chronic—traumatic cases of course excluded. The condition is therefore described in the next section. In the very rare cases in which focal meningitis is acute, the symptoms are, for the most part, similar to those of the chronic form, the chief difference being in their course.

Chronic Meningitis.—The symptoms of chronic meningitis consist chiefly in local disturbance of function; the symptoms outside the nervous system, so conspicuous in most cases of acute meningitis, are, as a rule, absent in the chronic form. In the adult the symptoms vary much according to the cause and nature of the inflammation. In the *chronic alcoholic meningitis*, which affects chiefly the convexity over both hemispheres, the chief symptoms are headache, moderate in degree and sometimes absent, slight delirium, mental failure, and slight optic neuritis. The malady always runs a very chronic course, and its symptoms are often overshadowed by those of some other of the effects of alcoholism, such as liver disease or multiple neuritis. The symptoms may subside if the cause of the morbid state can be arrested. It is probable that they are in part due to the action of the alcohol on the brain substance.

Chronic syphilitic meningitis in the adult is generally local. It usually occurs chiefly in the vicinity of a syphilitic growth, and its chief effect is to extend the symptoms of the latter over a wider area than corresponds to the actual tumour. Thus in the case of a syphiloma growing into the hemisphere from the outer side of the crus, the effects of this were accompanied by palsy of the fifth nerve on the same side in consequence of syphilitic meningitis extending from the tumour. Sometimes such meningitis exists apart from any distinct growth, and may then extend widely, even sheathing the whole base with its products. There is always a considerable formation of tissue thickening the pia-arachnoid, sometimes to such an extent as to suggest a diffuse growth. The seat of the disease may be at the base or the convexity; in either situation focal symptoms are produced, scarcely distinguishable in the more chronic cases from those of a syphilitic growth except by the slighter degree of optic neuritis, the slighter indications of compression of the parts affected, and the less gradual onset. The side of the medulla is an occasional seat of this inflammation, which is one of the most common causes of the conjoint palsy of one half of the tongue, side of the palate, and vocal cord (see p. 307). Another occasional seat is the neighbourhood of the motor convolutions producing unilateral fits, and local pain and tenderness. It is probable that focal inflammation in adults is generally syphilitic in nature, the traumatic form of course excepted.*

In such cases it is often useless and even cruel to suggest the probable cause of the malady. It is unlikely that by such questions syphilis can be absolutely *excluded*, and unless it can be excluded the employment of antisyphilitic treatment in such a case is the first duty of the practitioner. Moreover, if syphilis can be excluded, the treatment suitable for syphilis (mercury) remains the most promising for a simple inflammation.

Chronic infantile meningitis, which is also in some cases associated with syphilis, causes symptoms that depend on the special proclivity of the inflammation to affect the posterior fossa of the base. The most important and characteristic symptom is retraction of the head. In a valuable paper on these cases† Drs. Gee and Barlow point out that the onset of this symptom is sometimes sudden, sometimes gradual; when sudden it is occasionally attended by other symptoms,—fever, vomiting, rigidity of the limbs, convulsions. I have known general convulsions to precede for a week the onset of the retraction. The holding back of the head is due to a tonic contraction in the muscles, and becomes greater when the child is made to sit up. It varies in degree at different times, and is occasionally intermittent. When the condition has become established, it is often attended by rigidity of the limbs, sometimes by epileptiform convulsions. Palsy is rare, but strabismus and nystagmus are sometimes observed.‡ Occasionally hydrocephalic enlargement of the head follows after a time. The symptoms continue for a period that varies from a month to a year and a half, and may end in recovery or death. After death the signs of chronic meningitis are always found, chiefly in the posterior fossa of the base; generally lymph glues together the medulla and cerebellum, the openings from the fourth ventricle are closed by the lymph, and the ventricles are distended. There may also be slight inflammation in the membranes of the spinal cord, and this, as I have seen, without special symptoms of spinal meningitis. In some cases such retraction of the head dates from the time of birth, and it is highly probable that in these cases there is hæmorrhage into the membranes about the medulla, a proved lesion depending generally on laceration of the cerebellum (see "Meningeal Hæmorrhage"). The retraction of the head must be distinguished from mere inability to support the head due to weakness of the muscles. I have, however, known such inability to follow the retraction in cases in which the symptom dated from birth.

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* The following case is probably an instance of it. A married lady, a month after her confinement, suffered for a week or two from neuralgic pain about the right temple and eye, and then became feverish; one or two convulsions occurred of uncertain character, and left hemiplegia developed rapidly, the paralysis of arm and leg becoming almost complete in the course of a few days. There was no optic neuritis. Iodide of potassium was given and mercury rubbed in, &c.; as soon as the influence of the drugs became established the symptoms began to lessen, and they passed away entirely in the course of a few weeks. In this case the evidence of local meningitis afforded by the symptoms and their mode of onset was conclusive; and although the effect of the drugs employed is not certain evidence of the nature of the lesion, it gave some support to the opinion, based on the limited extent of the disease, that it was of syphilitic origin.

† "On the Cervical Opisthotonos of Infants," 'St. Bartholomew's Hospital Reports,' vol. xiv, 1878, p. 22. The occasional relation to syphilis, noted by them, is confirmed by Money, 'Treatment of Disease in Children,' 1887, p. 456.

‡ Money (loc. cit.), who found also persistent slight pyrexia in one case.

Pathology.—The pia-arachnoid differs from most other membranes that enclose viscera, in its separation into two layers. Nevertheless it is commonly regarded as a serous membrane, and it presents some analogies to other serous membranes in its pathological liability, but also wide differences from them. Like the pleura, it is prone to spontaneous inflammation; but the most common cause of primary pleurisy, exposure to cold, seems to have little influence in exciting meningitis. It is the seat of specific processes more frequently than any other serous membrane, and this, together with its liability to suffer in states of blood-poisoning, must be regarded as its chief pathological characteristic. The process of inflammation also presents some peculiarities in the cerebral membrane. The tendency to the formation of lymph is smaller, and of pus is greater, than in the case of the pleura or the pericardium. Embolic processes may play a part in the generation of some forms of septicæmic inflammation, but it is probable that the circulation of septic matter in the blood, not necessarily organisms, suffices to excite in the membranes the inflammation to which they are prone.

The acute simple or purulent meningitis that occurs in children and adults, as an apparently primary affection, seems to be inseparable from the sporadic form of cerebro-spinal meningitis. Although spinal symptoms, beyond some retraction of the head, may be absent, purulent inflammation extends, in most cases, down the membranes of the cord. The probable pathology of these cases is considered in the section on the epidemic form.

The relation between the morbid process and the symptoms that reveal its presence is still, in part, obscure. The affection of the cranial nerves is usually due to their actual inflammation, indications of which may be found in the changes of interstitial tissue, and in the degeneration of the nerve-fibres. With regard to the character of the symptoms, we are doubtless safe in recognising, with the older writers, the early stage of over-action as the result of irritation of the nerve-elements by the inflammation of the enclosing membranes. In some cases the excitement of function, general so far as the meningitic influence is concerned, seems to be determined in its special form by predisposition, or by coincident influences. Hence it is that in specially disposed subjects the early symptoms may present characters definitely hysterical in their character. But the process of irritation may at first arrest function instead of exciting its activity. In the early stage of meningitis there is sometimes complete hemiplegia, and I have even known the inflammation to be apparently slighter over the hemisphere the function of which was thus arrested, than over the other. We can only explain such an arrest of function as the result of irritative inhibition, and must remember that pus may be abundant in the pia mater and yet not penetrate the centre. (See "Spinal Cord," in vol. i, p. 272.) Hence the frequent disproportion between the amount of inflammation and the strange fact that extensive purulent meningitis may run a latent course. For the depression of function in the later stage there are two possible mechanisms: first, the ventricular effusion and compression of the brain; and second, the greater damage of nerve-elements that have been previously excited.* Whether the tubercles precede inflammation or excite any symptoms by their presence is as yet an undecided problem.

Considerable paralysis of the cranial nerves is no doubt always due to the affection of these nerves at the base of the brain, with the exception of palsy of the face and tongue, which may be considerable in degree when of cortical origin. Thus I have seen paralysis of all parts of the face associated with paralysis of the limbs on the same side, but the palsy of the face involved voluntary and not emotional movements—conclusive evidence that it was not due to an affection of the nerve, and it soon becomes confined to the lower part. Spasmodic strabismus and inequality of pupil may also probably be due to the affection of the cortex, as well as to that of the nerve-trunks. Rigidity of muscles is a result of irritation, either of the cortex, crus, or pons; the retraction of the head is due to inflammation about the pons and medulla,—not, as is often said, to the ventricular effusion. Whether any symptoms, in the occular muscles or elsewhere, are due to inflammatory irritation of the corpora quadrigemina, or of the grey matter lining the third ventricle, is uncertain. The nerve nuclei are sometimes damaged by hæmorrhage, &c., from interference with those arteries that pass through the posterior perforated spot.

DIAGNOSIS.—The diagnosis of meningitis rests on the presence of such symptoms as indicate organic disease at the surface of the brain, on the development of these in an acute or subacute, but not sudden manner, and on the presence of the general symptoms of inflammation, and of a probable cause. Of the cerebral symptoms, those of local character are the most conclusive, but often of least actual diagnostic importance, since they occur comparatively late in the course of the affection; and although it is now and then impossible to recognise the disease until they appear, they more often serve to confirm than to establish the diagnosis. These general cerebral symptoms are significant by their degree and combination, since most of them may be produced by processes commencing outside the nervous system. The significance of the headache depends on its persistent intensity; of the delirium, on its co-existence with headache; of vomiting, on its causeless recurring character; of general convulsions, on their association with the other symptoms; of frequency of pulse, on its combination with the pyrexia that usually accelerates the action of the heart. It is in the early stage, when these general symptoms alone exist, that the chief diagnostic errors occur; but when the inflammation is at the convexity, this difficulty may persist until late in the disease. A child of five had headache, vomiting, and drowsiness for a fortnight, then became comatose, and died in a few hours, even the optic discs being normal. Among important local symptoms are the inequality of pupil; strabismus, even if transient; nystagmus; retraction of the head, and weakness in the face. The ophthalmoscopic changes may also decide the diagnosis, chiefly in cases in which they occur early, or local symptoms develop late. In some obscure cases the occurrence of retention or incontinence of urine, or the readiness with which the skin blisters, gives sufficient weight to other symptoms to determine the diagnosis. In all cases the Protean character of the disease should be remembered, and above all the fact that there is no symptom of meningitis that is not sometimes absent. Headache is the most constant, rarely wanting, but cases have been known to run their course without any obtrusive pain; its absence, therefore, does not destroy the value of other symptoms that are of significance, and there is no other symptom (not excepting pyrexia) the absence of which is of much negative value. The presence or history of a possible cause of meningitis often assists the diagnosis by directing attention to symptoms that might otherwise be unnoticed. This is especially the case with injuries, ear disease, pyæmia, and phthisis. Any brain symptoms in the subjects of tubercular or scrofulous disease, even at a distance, should excite concern. Occasionally disease near the brain may render the diagnosis more difficult by obscuring, with its own obtrusive symptoms, those of its effect.

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* Rilliet and Barthez, Huguenin, &c.; also Wilks and Moxon, 'Path. Anat.,' 2nd ed., p. 210; Hilton Fagge, 'Princ. and Pract. of Med.,' ed. by Pye-Smith.

The existence of meningitis being recognised, the diagnosis of its seat depends chiefly on the character of the symptoms—whether they are such as to indicate disturbance of the convexity or of the base. The differences that depend upon locality have been already described. Sometimes the cause of the meningitis also helps the diagnosis of locality, since adjacent disease excites inflammation first in its own neighbourhood.

In determining the nature of the inflammation we are guided by its cause, its associations, its seat, the age of the patient, and the relative frequency of the several forms. Each of these points must be considered, not only in itself, but in relation to the others. The apparent cause often decides the question: from adjacent caries and in pyæmia the inflammation is almost certainly purulent; but if there is distant suppuration of scrofulous origin the inflammation may be either tubercular or purulent, and the former is more probable unless there are other signs of septicæmia. In pneumonia or acute specific diseases the inflammation may be either simple or purulent. If there are signs of phthisis or a tubercular family history, and no other cause can be detected, the meningitis is almost certainly tubercular. The discovery of tubercles of the choroid renders the nature of the inflammation certain. A boy, aged five, was seized with acute pain in the head on the twentieth day of typhoid fever (with characteristic eruption); this was followed by coma, inequality of pupil, deviation of head and eyes to the left, rigidity of the left limbs, and half-purposive movements of the right. I saw him on the fifth day after the onset of these symptoms, and found optic neuritis in the right eye only, with tubercles in each choroid,—a proof that the inflammation was tubercular, complicating the fever and not due to it. If the inflammation is at the base of the brain, and no cause is discoverable, the probability that it is tubercular is very great. If the inflammation is at the convexity the probability of its tubercular nature is considerable in childhood and youth, but in adult life such inflammation is probably not tubercular. Under twenty years of age there is a presumption, in the absence of other causal indications, that any meningitis is of tubercular origin; but over forty there is a presumption against this, which becomes greater as life advances, and is increased by a history of alcoholism or of syphilis. The special diagnosis of ventricular meningitis is practically impossible during life.

Differential Diagnosis.—Meningitis may be confounded with general diseases, and with other diseases of the nervous system. These errors occur chiefly with tubercular meningitis, because most other forms have an association with some cause which prevents a mistake, or else produce symptoms so acute and characteristic that there is neither time nor room for error. The following remarks on the differential diagnosis apply, however, also to the rare simple basal meningitis which, during life, cannot be distinguished with certainty from the tubercular form.

The error of mistaking an acute general disease for meningitis is more common than the opposite error. Typhoid fever and catarrhal febricula are the diseases with regard to which a mistake is most common; the former in older children and adults, the latter in young children. The error always arises from ascribing initial headache and subsequent delirium to cerebral disease in the presence of sufficient pyrexia to account for the symptoms, and from disregarding the relation between the two. When they are the result of a general disease, as Sir William Jenner long ago pointed out, the headache ceases when the delirium begins. In meningitis the headache continues, and co-exists with the delirium. In children, convulsions may occur at the onset of the general disease. In all cases, therefore, in which there is first headache and then delirium, and in which there is pyrexia sufficient to account for both, the presumption is strongly against meningitis; this should only be suspected when local cerebral symptoms appear, or if there be general cerebral symptoms such as optic neuritis, that do not occur during general diseases. The difficulty of the diagnosis between typhoid fever and tubercular meningitis is increased by the fact that the former is sometimes attended by constipation, and the latter by diarrhœa due to tubercular ulceration of the bowels, or by general abdominal tenderness from the formation of tubercles in the peritoneum. It is sometimes necessary to wait and watch the course of the symptoms before a confident opinion can be formed. As a rule, the pyrexia runs a more regular course in typhoid, and the pulse is more frequent and is seldom irregular. The optic neuritis that is occasionally associated with acute specific diseases does not accompany, but succeeds them. It should be remembered that convulsions which are partial, and begin locally, are local and not general cerebral symptoms. Avoidable error arises in most cases from the judgment being warped by the initial headache, and the practitioner omits to keep a look-out for other symptoms outside the nervous system. In consequence of this, gross mistakes are sometimes made.

Retraction of the head, resembling that which occurs in meningitis, is sometimes the result of rheumatism of the muscles at the back of the neck. Legroux asserts that in such cases there may be considerable fever,* but distinct cerebral symptoms are of course absent, and the muscles are more painful than in meningitis. Such muscular rigidity is said to be also produced, in some cases, by peripheral irritation, tender cervical glands, and abdominal disturbance.†

Among diseases of the nervous system, one that sometimes gives rise to considerable difficulty in diagnosis is intra-cranial tumour. A rapidly growing tumour, especially one that at first interferes little with function, may cause symptoms which develop so rapidly as to be easily mistaken for those of meningitis. This is the case sometimes with tubercular tumours, and with glioma of the pons, which may run an almost latent course until they have reached a considerable degree of intensity. The symptoms in the limbs often assist the diagnosis, since the loss of power is more often an early symptom in tumour than in meningitis, and it comes on gradually in the former, whereas early paralysis of the limbs in meningitis usually comes on suddenly from irritative inhibition. The ophthalmoscopic appearances are of great importance. A slight degree of neuritis may be due to either tumour or meningitis, but an intense neuritis, with considerable swelling and hæmorrhages, is practically conclusive of tumour. The neuritis of a rapidly growing tumour is usually intense, and when the apparances are slight at first, if the disc be watched for a few days, the course of the neuritis often decides the diagnosis. It must be remembered that from the absence of neuritis no conclusion can be drawn. In many cases, however, the question can be only decided from the course of the disease. If, after the first two weeks from the commencement, the symptoms continue, slowly increasing, and the patient does not become comatose, the diagnosis of tumour is almost certain. It must not be forgotten that meningitis often co-exists with tumour, by which it is excited, but the symptoms of tumour in these cases have usually preceded those of meningitis, and continue unchanged when the latter have passed away or lessened. When such meningitis is excited by some adequate exciting cause, as a fall, the fact that there is more than inflammation may only be shown by the persistence of some significant symptom after the meningitic disturbance has passed away.* A difficulty is met when tubercular tumours co-exist with tubercular meningitis, but the difficulty in all these cases is rather in the recognition of the tumour than in that of the meningitis.

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* Legroux, 'L'Encéphale,' 1885, No. 1.

† Money, 'Treatment of Disease in Children,' 1887, p. 457.

In some cases of meningeal hæmorrhage the symptoms have an acute, not sudden onset, and may closely resemble those of inflammation over the convexity; headache and delirium are conspicuous; and both diseases may follow an injury. This form of meningeal hæmorrhage is, however, very rare. The most important criterion is the absence of fever and the rapidity of course. Sudden symptoms in the course of meningitis do not lessen the probability of the diagnosis, because they may be due to a secondary vascular lesion.

A considerable difficulty in diagnosis is presented by some cases of disease of the ear, in which symptoms resembling meningitis occur and may prove fatal, although after death only thrombosis in a sinus can be discovered, and sometimes even that is absent. The central symptoms are for the most part general, and may be in part due to pyæmia; but strabismus has been observed, and the difficulty in diagnosis is increased by the fact that optic neuritis may occur and may even reach a considerable degree. It is doubtful whether in these cases a positive diagnosis is always possible.

The same difficulty presents itself in another form in cases of acute double otitis in children, which may be attended with intense pain in the head, vomiting, fever, delirium, giddiness, convulsions, and bilateral deafness. In most instances the labyrinth is chiefly affected, and a post-mortem examination at a later period has shown only symmetrical changes, sometimes limited to the cochlea and semicircular canals, with atrophy of the auditory nerve.* Such cases are constantly thought to be meningitis;† but it is probable that inflammation of the membranes never damages gravely the auditory nerves without the adjacent facial nerves.‡ The general cerebral symptoms alone scarcely warrant the diagnosis of secondary meningitis excited by the inflammation of the ears. It is probable, moreover, that in some of these cases there is optic neuritis, although there is no meningitis. Most practitioners have met with cases in which complete deafness and blindness came on in childhood with such acute cerebral symptoms, and in whom the ophthalmoscope reveals the form of optic nerve atrophy that succeeds inflammation.§ It is probable, on account of the age of the sufferers and the symmetry of the disease, that the affection is due to a blood-state, sometimes induced by cold, and it has been conjectured that this state is analogous to that which causes polio-myelitis. The cases present a very difficult diagnostic problem, and the difficulty is increased by the fact that the internal ear may be inflamed secondarily to meningitis. Such secondary otitis has been observed in cerebro-spinal meningitis, but it is very rare.‖

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* Thus characteristic and severe meningitis of the convexity, bilateral, followed a fall on an ice-covered hill-side. The patient recovered save for optic neuritis, which persisted without diminution, and there followed hemiplegia, evidence of a tumour of the right hemisphere, and death.

The condition of depressed cerebral function that was termed by Marshall Hall *hydrocephaloid*, and is apparently due to anæmia of the brain, may be confounded with tubercular meningitis. In this condition, which is almost confined to young children, there is somnolence and coma, with depressed fontanelle, and local symptoms are absent. It is said, indeed, that rigidity of the neck and strabismus have been observed, but there is some doubt as to the real nature of cases that present such symptoms. The cases are distinguished from meningitis by the depression of the fontanelle, and especially by the occurrence of the symptoms in the profound exhaustion that results from diarrhœa or loss of blood.

Of the general diseases of the nervous system there is only one that is liable to be confounded with meningitis, and especially with tubercular meningitis,—hysteria. This error is by no means rare, but occurs only in the cases in later childhood and youth, and especially in the female sex. Meningitis, especially tubercular, is far more frequently mistaken for hysteria than hysteria for meningitis. The former error is very common; it is usually due to the fact than when there exists the state of nervous system that underlies hysteria, pronounced hysterical symptoms are often developed during the early stage of tubercular meningitis. The case is diagnosed as one of hysteria, and when other symptoms develop, they are disregarded under the influence of the preconceived idea. Even when no hysterical symptoms attend the attack, if such have occurred in the previous history of the individual (as is often the case in tubercular girls), the assumption that the cerebral symptoms are due to hysteria is often made when there is not the slightest justification for it. Indeed, in not a few instances the fact that a girl is the subject of vague general cerebral symptoms is allowed to determine the diagnosis. The only way in which error can be avoided is to search and watch for symptoms of organic origin, and allow these the same weight as in a case in which there are no hysterical symptoms, or as in a patient in whom hysteria would not be expected. Pyrexia is of especial diagnostic value in these cases, and so also are convulsions beginning locally, and the ophthalmic symptoms. Strabismus in hysteria is always convergent and attended by spasmodic contraction of the pupils. Divergent strabismus, or inequality of pupil, or nystagmus, is certain evidence of organic disease, and as much so if it is transient as if it is permanent. Retention of urine may be due to hysteria, but incontinence never is. The significance of a tendency for the skin to blister has been mentioned; signs of trophic lesions of the skin should be carefully looked for in every doubtful case.*

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* Politzer, 'Diseases of the Ear,' Cassell's translation, 1883, p. 714.

† The facility with which this mistake may be made was pointed out by Votolini ('Monatschr. f. Ohrenheilk.,' 1870, Nos. 7 and 8) and Reichel ('Berl. kl. Wochenschr.,' 1870, Nos. 24 and 25).

‡ It has been assumed that the auditory nerves may be so damaged at the base as to cause complete deafness without any paralysis of the facial. This assumption rests on no evidence, and is most improbable, although no doubt, as already stated, the auditory nerves may suffer somewhat more readily than the facial in slight inflammation of the membranes.

§ A series of such cases has been recorded by Mr. Hutchinson, 'Ophth. Hosp. Rep.,' 1866.

‖ Lucae, 'Arch. f. Ohrenheilk.,' Bd. v, 1870, p. 188. The otitis is so rare, and so remarkable in its bilateral character, that it may possibly be a coincident effect of the blood-state.

An illustration of the difficulty that sometimes attends the diagnosis, and of the significance of pyrexia, was afforded by the case of a servant, aged twenty-two, who had had a child three years previously, and had suffered from some headache for six weeks. On account of slight indisposition, her mistress suspected another pregnancy, and, without telling the girl, sent for a medical man to see her. The girl was intensely annoyed, and refused to answer any questions. She went up to her room and fell, bruising her face. All the rest of the day she was dull and lethargic, occasionally, however, throwing her arms about, screaming, and complaining of pain in her head. The symptoms were ascribed to hysteria, but as she was no better next day she was brought to University College Hospital, and admitted. Her temperature was found to be 101°, and the bladder full, so that the catheter was used. For some days she continued dull in aspect and manner, sometimes answering when spoken to, sometimes not.

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* It should be remembered that these trophic disturbances are often not mentioned, or are even denied, by nurses, who fear that the sores may be ascribed to carelessness.

She complained of pain in the head and abdomen. One day she was childish, playing with a doll. The temperature, however, continued raised, varying from 102° to 99°. On the seventh day she became semi-comatose, and passed urine into the bed. On the ninth day she rather suddenly became dusky, with irregular breathing, and mucus in the chest. Death from respiratory failure being manifestly imminent, artificial respiration was employed, and by this, faradism to the chest wall from time to time, and food through a long catheter, she was kept alive for twenty-four hours, and died on the eighth day from the onset. Throughout there had been no symptoms in the limbs or cranial nerves. The post-mortem examination revealed general tuberculosis of lungs, peritoneum, and intestine, some small masses of yellow tubercle in the cerebral hemispheres, and meningitis of the base, the lymph being especially abundant about the pons and medulla, with opaque tubercular granulations.

The converse error, in which hysterical symptoms are regarded as meningitis, is far less common. The mistake is sometimes made in cases of hysterical sopor with the strong convergent strabismus, but the distinct spasmodic character of the latter is usually distinctive, and there is, as a rule, no alteration of temperature. The last point is also of diagnostic importance in the curious state of trance-like sleep that sometimes comes on in states of brain-exhaustion, usually in hysterical subjects, but in lads as well as girls. When this succeeds severe headache, as it often does, the difficulty of diagnosis may be great. But it should be remembered that it is rare for coma to come on early in the course of meningitis. Tetanus may be thought to exist in the cases of meningitis in which contracture involves the muscles of mastication, but the symptom soon passes off.

PROGNOSIS.—In every form of meningitis the prognosis is grave; it is least serious in the traumatic form, and in simple meningitis from adjacent disease it is most grave in the purulent form, recovery from which is practically unknown. Nevertheless I have twice known recovery from distinct symptoms of meningitis in post-puerperal septicæmia. In any form, if the stage of coma has been reached, death is all but certain.* But the patient has some small chance of recovery in simple meningitis, and perhaps (although still slighter) in tubercular meningitis; and, moreover, the very important fact must be borne in mind that the diagnosis between the two, and between these and meningitis secondary to obscure adjacent disease, is a matter of probability only, however high the probability may be.

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* A case of recovery from an illness resembling tubercular meningitis, in which the stage of coma was reached, and the child seemed for days to be on the point of death, is recorded by West ('Diseases of Infancy and Childhood,' 7th ed., p. 96). It is said, however, that the child became blind and afterwards recovered her sight, a feature which does not suggest tubercular meningitis.

Hence it is not right, in any case, to assert the *certainty* of a fatal issue.*

In all cases the most material prognostic indication is afforded by the course of the disease. The less acute the attack, the more chance of recovery the patient has. If, at the end of the third week from the onset, the patient has not passed into a state of coma, there is an appreciable diminution in the probability of death. The chance that death may be escaped is least of all when the coma comes on before the first week is over.

TREATMENT.—If the meningitis is due to adjacent disease, the treatment of this is of the first importance. If none is obtrusive, the ears should be carefully examined, since, as the case just mentioned shows, suppuration in the middle ear may excite meningitis even though no discharge has previously been noticed. A free exit should always be made for any collection of pus in the neighbourhood of the skull, and if there are any indications of the presence of pus in the tympanic cavity, the membrane should be incised. If no such cause can be discovered, the treatment must be directed to the diminution of the local inflammation and the counteraction of the general state on which it depends. Neither the seat nor the form of inflammation has much influence on the treatment. This has to be conducted in each case on the same general principles, variations being determined by individual differences much more than by pathological nature.

In no disease is perfect tranquillity of greater importance. The patient should be disturbed as little as possible, kept free from all excitement and all mental exertion; the room should be darkened if there be any intolerance of light, and in all cases it should be kept as quiet as possible. But mental depression should be avoided only less carefully than mental excitement. Sleep should be encouraged, and the patient roused only for the purpose of feeding. Light nutritious food should be given every three or four hours, and if the patient cannot be made to swallow, nutrient enemata should be administered. Stimulants are best withheld unless the state of the pulse urgently calls for them. The head should be high, but the shoulders also raised to avoid flexion of the neck and mechanical hindrance to the return of blood from the head.

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* As an illustration of this I may mention the case of a boy, five and a half years old, with tubercular family history, who, after five days of vague general indisposition, became feverish, with severe headache and double vision. He soon became so weak as to be unable to walk. I saw him after the cerebral symptoms had lasted for five days. The child appeared very ill; the temperature was 101°; the tongue was covered with a thick white fur, both sixth nerves were paralysed, and there was weakness of the left side of the face. There was no history of injury or discharge from the ear. The case was certainly one of meningitis, and it seemed highly probable that it was tubercular. Two days later, however, there was a sudden and copious discharge of pus from one ear, the cerebral symptoms rapidly disappeared, and the boy recovered perfectly.

Abstraction of blood, while undoubtedly in many cases a powerful agent in lessening local inflammation, is rarely desirable in meningitis. Exceptions to this rule are acute forms following injury, exposure to the sun, or excessive mental excitement, and inflammation for which no probable cause can be traced. In these, if the patient be strong and full-blooded, leeches may be applied behind the ears or to the temples in the early stage of the disease. During the late period abstraction of blood can do only harm. In strong children leeching has also been recommended, but the cases are few in which this is desirable. Most cases occur in weakly anæmic individuals in whom loss of blood reduces the strength of the inflammation less than it does the strength of the patient. Attempts may be made to keep the blood away from the head by warmth and mustard to the extremities, and by cold to the head. The hair should be cut short, or the head shaved. The application of cold should be continuous, either by an ice-bag (half filled, and containing no air, so that it may adjust itself to the head), or by a cap made of metal tubing, wound in a spiral, through which water may flow from a vessel above the level of the head to one on the floor.* This is a very convenient mode of applying cold, as effective as ice, and available when ice cannot be obtained.

Counter-irritation to the occiput is also sometimes distinctly useful. Mustard may be employed, but a blister is more effective. There is a natural reluctance to add the pain of a blister to the sufferings of the patient, but a blister certainly often lessens the intensity of the symptoms, and it is probable that the total amount of pain endured is at least not increased thereby. Counter-irritation over the whole scalp, by blister or irritating ointment, has sometimes been recommended, but it interferes with the application of cold, and it is doubtful whether its influence counterbalances this disadvantage.

Vomiting is best allayed by ice to the head and given by the mouth; and by two mustard plasters applied, one to the back of the neck, and the other to the epigastrium, or by the administration of cocaine to lessen the peripheral impulses from the mucous membrane, which act on the centre. The bowels should be opened with a freedom which must depend on the strength of the patient. Constipation is often difficulty to overcome, but is injurious, while purgation is one of the most effective derivatives. Headache is often lessened by ice, and sometimes by antipyrin; opium should be avoided.

Internal remedies must vary with the state of the patient. In all cases with elevation of temperature a diuretic is safe and often useful. Digitalis in small doses may be given if the pulse is irregular or unduly frequent in the early stage. When the patient was previously anæmic and the temperature is not very high, iron may be given in some non-irritating form, such as dialysed iron, or the citrate of iron and quinine, but the bowels must be carefully kept open.

Various drugs have been given with a view of influencing directly the process of inflammation, and each has been in turn extolled, but the only one which has obtained wide recognition as distinctly useful is mercury. With this opinion, my own experience fully agrees. It is naturally more often effective in simple than in tubercular inflammation, but it was used in each of the cases apparently of the latter form in which I have seen recovery take place. The patient should be brought slightly but distinctly under its influence, so as just to "touch the gums," as the phrase is, as quickly as may be, because only thus have we evidence that it is not excreted as fast as it enters the blood, and that it actually acts upon the tissues. Inunction is by far the best method of effecting this. A little mercurial ointment or oleate of mercury should be rubbed into the armpits and groins every four hours, or on the back of the neck or the scalp, until the effect is produced. Iodide of potassium is another remedy that has been much used, but the evidence of its value is slighter than is that of mercury. It may be given, however, combined with a tonic, after mercury has been discontinued.

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* Such caps can be obtained from Krohne and Sesemann.

When the temperature is very high, cold baths have been employed with some apparent temporary advantage, but the almost invariably fatal issue has not been retarded by the reduction of temperature. The same may be said of salicin, quinine, and antipyrin given for the same purpose.

In cases of septicæmic meningitis, what slender chance of benefit there may be is most likely to be obtained by the free administration of perchloride of iron, a drug which, more than any other, has seemed to me to have the power of saving life in septicæmia. This treatment was adopted in each of the cases of apparently septicæmic meningitis which recovered. In one such case there was severe headache and delirium, rigidity of the limbs on one side, and a temperature of 105·5°.

The treatment of acute ventricular meningitis does not differ from that of extra-cerebral meningitis—a fortunate circumstance, since the recognition of its situation is scarcely practicable during life.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

(CEREBRO-SPINAL FEVER.)

Acute meningitis sometimes occurs in epidemic form, many patients being attacked in a certain district in the course of a few months. The spinal membranes are affected as well as those within the skull, and hence the name by which the disease is known; but, as we have seen, other forms of inflammation may not be limited to the cerebral membranes. There is usually considerable fever, and often an eruption appears on the skin. Sporadic cases occur, closely resembling those of the epidemic form, and probably identical in nature.

The disease has been met with during the present century in various parts of Europe and in the United States. In certain countries, as Sweden and Germany,* it has been especially prevalent. In Sweden alone 4000 persons are said to have died from the disease between 1854 and 1860. A very severe epidemic occurred in France in 1837. In Great Britain the chief epidemics have occurred in Ireland, especially in 1846 and 1868; in England only slight outbreaks have been recorded, while in Scotland one small group of cases has been met with.† It has been doubted whether the isolated cases of acute severe and primary meningitis ought to be classed with the epidemic form, but they are not separable by any clinical or pathological features, and they have a distinct tendency to be multiple, which seems to associate them clearly with the epidemic form. Thus in the spring of 1887 four cases were admitted to University College Hospital, and two others came under my notice in other parts of London. Three of the cases occurred in the same street; and two others, though quite separate, developed within a quarter of a mile of each other. In Hamburg, where the disease has prevailed for ten years, most of the cases came from a few streets; and in an epidemic in Copenhagen most of the cases (185 in number) came from two centres, distant and unconnected. But cases that are quite solitary, although typical, are not rare.

CAUSES.—No age is exempt, but young persons under twenty suffer much more frequently than others, and in some epidemics children have been almost exclusively affected. Males are said to be attacked more frequently than females.‡ As it is a disease chiefly of temperate and cold countries, so it has prevailed chiefly in winter and spring, ceasing about July until the winter. No relation has been traced, as a rule, to local endemic influences, although some observers who have watched epidemics have thought that the incidence of the disease suggested some malarial or miasmatic influence, or that a relation could be traced to the amount of watery vapour in the air. But it has broken out simultaneously in places far apart, and has even been prevalent at the same time in Europe and America. It has occasionally preceded measles, subsiding temporarily, or continuing during the exanthem. Personal ill-health seems to have but little predisposing influence, but insanitary conditions and overcrowding apparently favour its occurrence, although it often occurs when there is no suspicion of this, and even in an epidemic the malady may not be worst where sanitation is worst. The relation to season has been explained by the overcrowding in the houses of the poor during cold weather.* In one epidemic in France the only persons attacked were soldiers crowded together in barracks; another in Ireland fell exclusively on the inmates of workhouses. Nevertheless, contagion seems to play a very small part in the production of the malady.† The attendants on the sick scarcely ever suffer. In an epidemic of thirty cases recently in Cologne no two cases came from the same house.‡ Nevertheless, instances have been observed in which an influence exciting the disease seemed to be conveyed by a third person. A series of such facts has been collected by Hirsch, and Frew believed that he traced such a connection between the cases in the small Scotch outbreak. Although even very young children are prone to suffer, a woman who had a mild attack, in the course of an epidemic, suckled her child throughout her three weeks' illness, and the child remained perfectly well.§ It is said that, in Ireland, each epidemic coincided with an outbreak of a similar malady in pigs and dogs.‖ One attack does not seem to confer immunity. A woman died from the disease during one epidemic who had passed through a similar attack five years before.¶ The period of incubation (when conditions for exact observation have been secured) has not exceeded five days.

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* It is curious that in 1862 Hirsch wrote, "As far as I know, Germany has been entirely spared, with one small exception;" and that, according to Ziemssen, since the first distinct outbreak in 1863 hardly six months have passed without an epidemic in some part of Germany.

† Frew, 'Glasgow Med. Journal,' 1884.

‡ Of 255 fatal cases in Stockholm, 149 were boys, 106 girls.

Symptoms.—The manifestations of the disease are in part those of the local inflammation from which it takes its name, in part they are those of a blood disease. In cases of moderate severity, malaise and discomfort in the head may precede the onset for a few hours or for two or three days, seldom for a longer time—one or two weeks.** Sometimes there is vomiting, or slight stiffness at the back of the neck, for a day or two before the acute symptoms come on. In other cases the onset is almost sudden. Vomiting and headache are usually the first pronounced symptoms; their commencement may be attended by a rigor: sometimes there is also pain in the back. The headache varies in its initial seat, but it often soon becomes general, and is always severe; it is constant, but there are from time to time intense exacerbations. Often the headache is accompanied by giddiness, and, as in other forms of meningitis, by intolerance of light and sound. Delirium in many cases is quickly added to the headache; it may be quiet or violent, but it soon gives place to somnolence and stupor, from which the paroxysms of violent pain may at times rouse the patient. Severe pain in the back is generally soon added to the pain in the head; it is felt chiefly in the neck and loins, and often referred to the neighbourhood of the spine rather than to the vertebral column itself. Sometimes it is severe in the sacrum. It is increased by movement, and may radiate around the trunk or into the limbs; it is sometimes severe in the knee-joints. Whether there is pain in the back or not, rigidity in the muscles of the neck is almost invariable, and in those of the back is frequent. This gives rise to the characteristic retraction of the head, usually moderate in degree, sometimes so great as to cause the neck to be at right angles to the rest of the spinal column. Often the contraction seems to be chiefly in the deeper muscles. Any attempt to flex the neck gives rise to pain. The retraction of the head is usually an early symptom, although it is not often present at the actual onset: sometimes it is delayed until the later stage of the disease. The rigidity of the rest of the spinal muscles is generally merely enough to prevent bending of the trunk, but it is occasionally so great as to cause opisthotonos. The limbs also are sometimes rigid; the legs are often drawn up, and the abdomen is retracted. Rarely there has been slight trismus. General hyperæsthesia of the skin is a frequent symptom and of some diagnostic importance. Convulsions occasionally occur at the onset of the malady or during its course; they are usually general, sometimes unilateral or local.

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* Medin, 'Nord. Med. Ark.,' 1880, and 'Deut. med. Wochenschr.,' 1881, Nos. 41, 42.

† Common exposure to causal influence may readily be mistaken for contagion, and many alleged instances are not free from this source of fallacy. Cf. Oebeke, 'Berlin kl. Wochenschrift,' 1891.

‡ Leichtenstern, 'Deut. med. Wochenschr.,' 1885, No. 31.

§ Rzadowski, 'Virchow's Jahresb.,' 1879, ii, 5.

‖ Fagge, 'Principles and Pract. of Med.,' on the authority of Ferguson, Vet. Off. to the Privy Council of Ireland. Epidemic cerebro-spinal meningitis has also occurred in horses; an outbreak of 37 cases was observed by Ackermann ('Virchow's Jahresb.,' 1880, i, 701).

¶ 'Virchow's Jahresb.," 1879, ii, 5.

** Sabarth, 'Breslauer Arzt. Zeitsch.,' 1879, No. 18.

The face is often pale, and has a shrunken aspect. The pulse varies much in frequency; in some cases it is not much above the normal, while in others it rises to 120, 140, or more. It is not often infrequent. The temperature is almost always raised, and it is generally considerably raised, often reaching 104°, 105°, or 106°, the greatest amount of pyrexia being noted towards the termination of the disease. Both the temperature and pulse-frequency present irregular variations, sometimes together, more often without any correspondence. The bowels are generally confined; the spleen is rarely enlarged. The amount of urine has been sometimes increased, and albumen is occasionally present; sugar rarely.

Eruptions on the skin are an important feature of the disease; they occur both in epidemics and in sporadic cases, but are more frequent in the former, although they vary much in different outbreaks. Various forms have been met with—erythema, herpes, urticaria, purpura; but the most significant are herpes and the purpuric spots. The latter are met with in a large proportion of the more severe cases, and are occasionally present in attacks of moderate severity.

They vary in seat, and are sometimes chiefly on the lower legs or forearms; in the most intense cases they may coalesce so as to give rise to dark diffuse extravasation into the skin over a considerable area. Herpes is very variable, but is common in some epidemics; in one outbreak of 29 cases herpes was present in 26 (Leichtenstern). In another, of 32 cases, only 6 developed herpes (v. Sydow). It usually begins on the lips, and may spread to other parts of the face, but has been seen on the ears and sometimes on the limbs. These eruptions frequently present bilateral symmetry, and the several forms are sometimes conjoined. They usually appear after the disease has lasted three or four days. Occasionally there is intense conjunctivitis, and even ulceration of the cornea.

Paralysis of cranial nerves, strabismus, inequality of pupil, &c., may be met with in this as in other forms of meningitis. The pupils are dilated, sometimes after initial contraction, and may be unequal. Deviation of the eyes to one side has been occasionally observed. Optic neuritis is common in cases that last for more than four or five days, and may cause permanent loss of sight if the patient recovers. In the Copenhagen outbreak it was found in one quarter of the cases. Palsy of the limbs is not common; when complete hemiplegia occurs early it is often due to irritative inhibition, but sometimes at a later period it is due to a focus of more intense inflammation over the motor region.* Occasionally there is paralysis of part of one side from this cause. In rare instances the symptoms of the spinal meningitis predominate over those of the intra-cranial inflammation, and complete paraplegia may result.† Myotatic irritability is usually lost in the legs in such cases, and sometimes towards the close in others, in which spinal symptoms are not specially marked.‡

Among the occasional effects of the disease, deafness is of especial importance. It seems commonly to depend, not on damage to the auditory nerve at the base of the brain§ or to the medulla, but either on extension of inflammation from the membranes to the labyrinth and middle ear, or on a simultaneous inflammation of the internal ear. The inflammation of the middle ear is shown, not only by the interference with conduction, but also by injection of the membrana tympani, and sometimes by the discharge of pus through it; it may succeed that of the labyrinth, or may be simultaneous and apparently due to the same cause.

The delirium that attends the early stage of the disease soon gives place to a condition of stupor, deepening to coma. The period at which this comes on varies according to the severity of the case; the delirium may last a few hours only, and the patient may become comatose before the end of the first day, or the coma may only come on at the end of a week. In cases that terminate fatally the coma is attended by signs of failure of the heart, and by sighing or irregular breathing, and the temperature may fall, or may rise to an extreme degree.

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* An instance is recorded by Charlewood Turner, 'Path. Trans.,' 1884.

† Strümpell, 'Deut. Arch. f. kl. Med.,' Bd. xxx.

‡ Carrington, 'Path. Trans.,' 1884, xxxv, pp. 54, 55; Leichtenstern, loc. cit.

§ See note on p. 353. Infiltration of the sheath of the auditory nerve with pus has been found when no deafness was noted, and there has generally been evidence of suppuration in the labyrinth when there has been absolute deafness.

The severity of the spinal symptoms, sometimes great, is more often moderate, and even transient or recurrent. Rarely the inflammation becomes severe at one spot, as in a case in which rigidity became extreme in the muscles of one shoulder, which afterwards wasted (Broadbent). Occasionally, in subacute cases, the spinal and cerebral symptoms seem to alternate.

The variations in the severity of the disease are accompanied by corresponding variations in duration. In the most acute cases the patient quickly becomes comatose, and dies at the end of one or two days, sometimes even in five or six hours from the onset. The acute form has been termed "fulminant." On the other hand, slight cases sometimes occur, in which the illness is trifling, and the symptoms consist in headache, pain in the back, and slight rigidity of the neck muscles—a form that has been termed, somewhat inaptly, "abortive." A remarkable epidemic occurred at a village in Lincolnshire a few years ago, in which all the cases were of this form. As in the case of other epidemic diseases, the severe cases are most frequent at the commencement, and the slight cases during the decline of the epidemic. The most acute cases are often attended by extensive cutaneous extravasation, and death seems to be due rather to the blood-change than to the meningeal inflammation. In the less severe form death generally results from asthenia, increased often by bed-sores. In cases that recover, the patient begins to mend some time during the second week. The mortality has varied, in different epidemics, from 20 to 80 per cent. Of 185 cases in an outbreak in Copenhagen about one half (49 per cent.) died. In sporadic cases it is also very high.

Besides the varieties that depend on severity, other forms have been occasionally met with. The fever has been observed to distinctly intermit, somewhat after the type of a quotidian or tertian ague, but with much less regular variations in the temperature than are presented by true intermittent fever. Other cases, in which the fever and a low asthenic state continue for a considerable time, have been described as a "typhoid" form.

Among other rare complications may be mentioned tonsillitis, multiple arthritis (sometimes suppurative),* endocarditis, and pericarditis. Abundant albuminuria has been associated with collections of the specific organisms in the glomeruli of the kidney. Combinations of cerebro-spinal meningitis with other maladies, such as scarlet fever, have also been described; it is doubtful whether the combination has been more than a coincidence.

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* Medin, loc. cit.

The process of recovery is generally very slow, and is occasionally interrupted by a distinct relapse. In one case of moderate severity, death, at the end of three months, seemed due to a secondary cerebral abscess. Headache persists for a long time, and a lasting liability to headache may succeed the disease. Of the sequelæ of the disease, the most important is the deafness from inflammation of the ear. It may be complete, and, occurring as it often does in young children, it may cause a loss of any power of speech that has been acquired, and permanent deaf-muteness. In some countries an epidemic of this disease has added enormously to the proportion of the population who were deaf and dumb. With the deafness there is often, for a time, a difficulty in maintaining equilibrium, due, no doubt, to the damage to the semicircular canals. It gradually passes away as compensation for the loss becomes established.* Noises in the ears may also persist for a long time. Another consequence occasionally observed is chronic internal hydrocephalus, which may cause its characteristic symptoms some weeks or months after the primary disease. It probably depends, in some cases at least, on its common cause, closure of the openings of the fourth ventricle, but it may sometimes be due to inflammation of the lining membrane of the ventricles (Merkel). An excess of fluid is sometimes found outside the cord. Mental change may persist for a time, but gradually passes away.

Pathological Anatomy.—In the most acute "fulminant" cases there may be only that transudation of hæmatin into the fluids of the body and the rapid decomposition which attend all intense toxæmic states, together with congestion of the membranes; in these the microscope may show collections of lymphoid cells along the vessels, or red corpuscles infiltrating the tissue. In cases of less rapid course there is intense hyperæmia of the pia mater of the brain and cord, with opacity and exudation of lymph, and in most cases that have lasted more than three or four days there is distinct formation of pus, which may accumulate in the subarachnoid space. It is more abundant over the posterior than over the anterior surface of the cord. The dura mater of the brain is little affected, but there are commonly some signs of inflammation on that of the cord. The ventricles of the brain may contain turbid fluid, sometimes pus, and their lining membrane may present signs of inflammation. Thrombosis in sinuses is rare. The substance of the brain may be pale, or may contain small hæmorrhages or points of softening, or small collections of pus. The spinal cord may also be inflamed and softened in places. The lesions in other organs of the body are such as result from other acute febrile blood diseases. The spleen and follicular glands of the intestinal canal may be found enlarged, but the enlargement of the spleen is slight and has seldom been detected during life. The kidneys are sometimes in a state of acute parenchymatous inflammation, as in other acute general diseases. The lungs often present signs of congestion, and endocarditis has been met with.

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* Moos, 'Meningitis cerebro-spinalis epidemica, &c.,' Heidelberg, 1881.

PATHOLOGY.—All the facts of the disease point to the existence of a specific poison acting on the blood, and through the blood exciting the local inflammation. It is, however, to the latter that the symptoms are chiefly due in all save the most intense cases. But of the nature of the influence which causes the disease, and the way it spreads, little is known. It is clear that children possess a special liability, which cannot be explained, as in so many specific diseases, by the mere absence of the protection afforded by a previous attack. The very slight part played by personal intercourse in the transmission of the disease has been already mentioned. The manner in which disconnected cases appear about the same time has suggested an analogy to influenza, but a still closer analogy exists between this disease and some forms of pneumonia, with which it is not unfrequently associated. The occurrence of herpes of the lips in each disease is a small but noteworthy point of resemblance. Pneumonia has been observed to be especially frequent at the time of epidemics of meningitis, and often co-exists with this disease. In such cases, either the specific organism associated with pneumonia (discovered by Fraenkel) is always to be found in both the lungs and the meningeal exudation, or else an organism of closely allied form, which is also present when there is no pneumonia. It usually presents the aspect, first noted by Leyden, of a diplococcus of oval or lanceolate outline.[*] It has been cultivated, but only above 86° F., and best between 95° and 99°. Inoculated, they kill quickly by blood-infection. These organisms are met with alike in the epidemic and the sporadic cases, but their source is quite obscure. Even in cases of very acute course and purulent character, with abundant diplococci, there may be no discoverable source of infection.

The organisms have been found chiefly in the lymph-spaces of the pia-arachnoid, penetrating the substance of the nerve-centres only in connection with aggregations on the surface.[†]

DIAGNOSIS.—The symptoms of meningitis, the severe headache and coincident delirium, the retraction of the head, the cutaneous hyperæsthesia, &c., are generally sufficiently distinct to prevent any confusion of the disease with other febrile maladies. The pain in the back, vomiting, and headache may suggest smallpox, but in cases in which the pain in the back is severe, the muscular rigidity usually soon manifests itself. It is said that in some cases of typhoid fever there is hyperæsthesia of the skin and some tenderness of the muscles of the neck (Leyden), but these symptoms quickly lessen, whereas in meningitis they increase. The herpes of the face may help to distinguish the disease from the continued fevers. In tetanus the opisthotonic spasm is generally secondary to trismus, which is scarcely ever an early symptom in meningitis.

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[*] Leyden, 'Cent. f. kl. Med.,' 1883; Eberth, 'Deut. Arch. f. kl. Med.,' xiii, p. 1; Nauwerck, 'Deut. Arch. f. kl. Med.,' xxix, p. 1; Senger, 'Arch. f. exp. Path.,' xx, 389; Cornil et Babes, 'Les Bactéries,' Paris, 1886, p. 446. See also Ziemssen, 'Handbuch der spec. Path.,' &c.,' ii, Th. 2, and Leichtenstern, loc. cit.

[†] Hoche, 'Psych. Vereins,' Karlsruhe, Nov., 1892.

Uræmia may cause muscular rigidity, convulsions, and coma, thus occasionally giving rise to symptoms somewhat like those of cerebro-spinal meningitis (Murchison), but the temperature is normal, and other symptoms of each malady are usually recognisable. It must be remembered that in children retraction of the head may occur from rheumatic affection of the muscles and other causes. It may also be produced by various morbid processes in the neighbourhood of the medulla; thus an aneurism on each vertebral artery (due to incomplete closure by embolism), and accompanied by optic neuritis, has been mistaken for this disease.

The greatest difficulty is the distinction of this from other forms of meningitis, and the question whether sporadic cases are to be regarded as examples of the disease. In other forms of cerebral meningitis spinal symptoms are seldom conspicuous, but it must be remembered that, in the epidemic form, there may not be more than retraction of the head. In tubercular meningitis the onset is more insidious, and coma is a rather late symptom, while a predisposition can usually be traced. The occurrence of the skin eruptions, the prevalence of the disease in an epidemic form, or associated with pneumonia, give material help to the diagnosis. The identification of isolated cases is a subject on which opinions differ, but it will probably be settled by the microscope. My own belief is that cases of acute severe rapidly fatal inflammation of the cerebral and spinal membranes are not separable from the epidemic disease, and the opinion is supported by the frequency with which pneumonia co-exists. The group of cases referred to on p. 359, of which three came from one street within a few weeks, must be regarded as belonging to the epidemic form, but these, and the other cases that occurred at the same time elsewhere in London, differed in no respect from the isolated cases that are met with every year. In at least one case of this group there was pneumonia.

Lastly, when the disease has been epidemic, some of its symptoms have been produced by the influence of fear, in what has been termed "meningitophobia," and the distinction of this from the "abortive form" has sometimes been difficult. The absence of fever and of definite objective symptoms usually suffices.

PROGNOSIS.—The disease is most serious, except in the slightest form, but even in this a benign course cannot be counted on with certainty. Recovery can scarcely be looked for if coma comes on before the fifth day. The prognosis in different epidemics must be influenced by the prevailing character of the disease; it is, as a rule, decidedly worse in the isolated cases than it is in epidemics. It is said to be least grave between ten and fifteen years. If there is inflammation of the labyrinth, some degree of permanent deafness is probable. In cases in which hearing is lost, any return of the perception of sounds conducted through the bone is of favorable significance.

TREATMENT.—No agent has been proved to exert a specific influence on the disease. The treatment does not differ from that suitable to other forms of meningitis, but there is perhaps less to be hoped for from mercury. Among the other drugs that have been used, for the most part without distinct result, may be mentioned iodide of potassium, salicylate and benzoate of soda, quinine, digitalis, and chloral. Iodide of potassium, salicylate of soda, and antipyrin have, it is true, been credited with a beneficial influence in some cases. In cases that are not rapidly fatal, great care is needed to secure cleanliness and to avert bedsores. The patient should, if possible, lie on the side, or at least not on the back, being supported by a plank protected by padding or pillows, so that the spinal column is not the lowest part towards which blood gravitates. Although we are still without the means of counteracting directly the active element in the disease, we may reasonably hope that the exact knowledge of its nature which we now possess may bring such means, before long, within our reach. For the prevention of the disease, fresh air and the avoidance of overcrowding are the most important measures.

ORGANIC DISEASES OF THE BRAIN.

ANÆMIA OF THE BRAIN.

The blood within the brain is contained in arteries, capillaries, and veins. The functional condition of the brain depends on the quantity and quality of the blood circulating in its capillaries, and it is to this that the special symptoms are related, and the effects thus produced are for the most part the same. Deficiency in the quality of the blood supplied to the brain is always of gradual occurrence, and affects the whole brain; deficiency in quantity of the circulating blood may affect the whole brain or part only, and it may be sudden or gradual in its production.

Causes.—*General cerebral anæmia* may be due to the following causes:—(1) It may be a part of systemic anæmia—defect in quantity or quality of the whole blood, due to various causes, as hæmorrhage, exhausting discharges, or defective blood-nutrition. (2) The supply of blood to the brain may be deficient, the quantity of blood in the body being normal. This may be due to cardiac weakness, or to causes acting through the nervous system on the heart, as in swooning. In systemic anæmia the lessened cardiac power increases the cerebral deficiency. Whatever lessens the amount of blood discharged from the heart, such as aortic obstruction or mitral disease, may be a cause of cerebral anæmia. Pressure on the vessels conveying the blood to the head, as by an aortic aneurism, has a similar effect. Unequal distribution of the systemic blood is another cause. The intestinal vessels, if dilated, are capable of containing a large part of the blood of the body, and the effect of their engorgement is often seen after paracentesis abdominis, and in the fainting that may attend diarrhœa. The effect of each cause is increased by the action of gravitation in the erect posture. (3) Cerebral anæmia has been supposed to occur during the exhaustion after the acute stage of febrile diseases, and to be the cause of certain cerebral symptoms that may attend this stage. The mechanism is, however, uncertain, since we have learned that such symptoms may be an after-effect of the organised virus of the disease, or a consequence of some toxic agent produced by the virus. (4) The capacity of the cerebral capillaries may be diminished by pressure on the brain, exerted by effusions of fluid (hydrocephalus), of blood (in cerebral and meningeal hæmorrhage), or by growths within the skull. Symptoms are produced especially when the mechanism acts rapidly.

Partial cerebral anæmia is due to some obstruction to the passage of the blood through the vessels. To be permanently efficient such obstruction must be situated beyond the circle of Willis. Ligature of one carotid causes immediate symptoms of cerebral anæmia, but permanent symptoms are not frequent. Pressure on, or disease of, one carotid, for the same reason, rarely gives rise to symptoms. Obstruction in certain arteries of the brain may cause local anæmia, sudden or gradual, temporary or permanent, according to its cause. Such obstruction may be due to narrowing of the calibre of the vessel by atheromatous changes in its wall, by syphilitic disease, or possibly by spasm of its muscular coat; but the mechanism of vaso-motor spasm has been extensively invoked to explain symptoms, merely because the origin of these was obscure. Complete anæmia may be due to actual occlusion by embolism or thrombosis; it constitutes the first stage of local softening, which supervenes after twelve or twenty-four hours. When the closure is imperfect, symptoms of less intensity result. The pressure-effects of an intruding substance within the skull (tumour, or clot) act most intensely in, and may influence only, one region of the brain.

PATHOLOGICAL ANATOMY.—The principal anatomical character of cerebral anæmia is pallor of the brain, observable chiefly in the paler tint of the cortical substance, and the diminished number of red spots in the white substance. The pallor may be partial or general. But pallor of the brain after death does not by any means necessarily show that anæmia existed during life. The amount of blood in the brain depends chiefly on the mode of death. The membranes are usually pale in anæmia, but in some cases of partial anæmia they are hyperæmic. In general cerebral anæmia, effusion of serum in the meshes of the pia mater and between the convolutions may be found, and it is even said that the brain-substance may be œdematous, and the nerve-cells changed in aspect, unduly translucent, or unduly granular. The walls of the minute vessels may also be found degenerated, thickened and homogeneous in appearance, a condition which may aid in the interference with function.* Partial cerebral anæmia, if absolute, is the first stage of softening, in connection with which its features are described.

SYMPTOMS.—The symptoms of this condition vary according as the anæmia is suddenly or slowly produced, and as it is general or partial. (1) In *sudden* general anæmia of the brain the sufferer feels drowsy; the special senses are dulled; noises in the ears and vertigo are complained of; the pupils are at first contracted; sight may fail; muscular power is weakened; respiration is sighing; the skin is pale, cold, and moist; nausea is common; and headache is rare. If the anæmia is more intense, consciousness is lost, there is universal paralysis; and general convulsions may occur, epileptiform in character, these being especially frequent in sudden extensive loss of blood in strong subjects. Nystagmus is sometimes observed. The pupils dilate,* and the coma may deepen to death. The loss of sight in cases which recover may persist as permanent amaurosis; but this is probably due to an affection of the retina. In syncope the patient may lose his sight when consciousness persists, or may remain blind for some minutes after the mental functions are restored.

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* Knoll, 'Wien. med. Wochenschrift,' 1885, No. 51.

(2) When general anæmia of the brain is *slowly* produced, the state of the cerebral functions is usually that of "irritable weakness." Their action is imperfect in degree, but may be excited with undue facility. There is mental dulness and drowsiness; sometimes, however, insomnia is troublesome. There are often hallucinations of the special senses, and it is said that maniacal attacks or melancholic depression may occur. Delirium is common in severe cases, as in some forms of imperfect blood-nutrition, the so-called "inanition delirium." Headache, usually general, is a common symptom; it is often neuralgic in character, and its dependence on this cause is shown by its relief in the recumbent posture, and (less surely) its removal by alcohol. Sensory hyperæsthesia, tinnitus aurium (especially the pulsating variety), muscæ volitantes, and vertigo are frequent. Sometimes the sight is dim, or there may be deafness, especially in the upright posture. Convulsions are rare, but muscular power is generally deficient. All these phenomena are more marked in the erect than in the recumbent posture, especially when the erect posture is suddenly assumed. It has been remarked that some anæmic persons can think well only when lying down.

In young children, after exhausting discharges, as diarrhœa, symptoms referable to cerebral anæmia sometimes occur—somnolence and pallor, with a depressed fontanelle and contracted pupils; occasionally there is strabismus and even rigidity of the neck. The somnolence may deepen to coma, the pupils dilate, losing their sensitiveness to light, and death may occur. Such symptoms have been called *hydrocephaloid* (first by Marshall Hall), from the resemblance to those of acute hydrocephalus.

(3) Partial cerebral anæmia causes, if complete, loss of function in the affected area; if it is permanent, necrosis of the cerebral tissue results. If incomplete and sudden, there is temporary arrest of function. Ligature of one carotid, for instance, may cause transient weakness and numbness in the opposite half of the body. There may be at first an over-action of grey matter, causing, in certain regions, unilateral convulsions. If slowly developed, as by the disease of

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* The early contraction is probably due to partial irritation of the third-nerve centre, the subsequent dilatation to its paralysis (see Mayer and Pibram, 'Präger Zeitschrift f. Heilkunde,' Bd. v, p. 15).

arteries, pain and vertigo are common, with recurring symptoms, such as numbness, tingling, and weakness referred to the limbs, &c.

In all cases of long-continued cerebral anæmia, lasting damage to the nutrition of the brain may result. In the child, the development of the brain may be arrested, and mental defect may increase to permanent imbecility, such as is met with after convulsions. In the adult, loss of memory and of general mental power may be produced; they last for months, and sometimes do not pass away. A lady, aged thirty-seven, had a prolonged attack of vomiting, which kept her in bed for three months, in the course of which she became extremely pale, dull, and lethargic, and for a time did not speak. On recovering, she remembered nothing of her illness, and thought it had commenced the preceding day. Six months later her memory was good for events before the illness, but she had no recollection of any event after it, even for an hour, and exhibited no tendency to improve. In all varieties of chronic anæmia these symptoms are common, and sometimes take the form of chronic insanity. The simple failure usually passes away sooner than definite derangement, but often not until long after the blood-state has improved.

Pathology.—The symptoms are, as already stated, dependent mainly on the defective quantity and quality of the blood circulating in the brain. Some effect has been ascribed to the diminution in the blood-pressure to which the nerve-elements are ordinarily exposed (Burrows)—a mechanical influence, of which the possibility cannot be denied and the probability cannot be proved. The precise cerebral disturbance, on which the symptoms directly depend, is uncertain. Doubtless the cortex is the first to suffer in function, and the mental symptoms are thus produced. The convulsions that result from sudden anæmia of the brain are usually ascribed to disturbance of centres in the medulla or pons, but the fact that compression of one carotid, which can affect only the cerebral hemisphere, has caused convulsion in the opposite half of the body, makes it more probable that the "discharge" is from the cerebral cortex. The convulsion is preceded by tingling in the whole of that side. That the nerve-cells should "discharge" when the blood-supply is arrested, is a fact of very great physiological interest as an indication of the reserve of force that must be stored up in the nerve-cells, and of the probability that sudden over-action is due to diminution of resistance to action, and not to an increase in the force-generating function of the cell. Latent energy may be liberated, but new force can scarcely be produced, under the influence of sudden anæmia. The disturbance of respiration, its sighing character, &c., are ascribed to the derangement of the respiratory centre in the medulla, but we cannot be sure that this is always the case. Such breathing has been observed to follow obstruction of the carotids. It must be remembered that our consciousness of dyspnœa, due to disturbance of the respiratory centre, must be produced through related cerebral centres, and that the respiratory functions must be widely represented in the hemispheres, so that they easily manifest emotional disturbance.

The permanent impairment of cerebral nutrition may be compared with the amaurosis which results from acute anæmia when no changes in the retina can be perceived. Analogy suggests a permanent shock to the nerve-elements and their nutrition.

Diagnosis.—The diagnosis is not difficult. It rests on the recognition, in a given case, of the causes of cerebral anæmia, and on the exclusion of graver maladies, such as organic cerebral disease. With the latter, it should be remembered, anæmia of the brain, local or general, often co-exists. Some symptoms of hyperæmia of the brain closely resemble those of anæmia. A common pathological state of imperfect blood-renewal within the capillaries probably exists in both conditions.

Prognosis.—The severity of the symptoms, and the extent to which the cause of the anæmia is amenable to treatment, and is of transient character, must determine the prognosis. As a rule this is favorable when there is no organic disease of heart, vessels, or brain. In the so-called "pernicious anæmia" the prognosis is, of course, far more grave. Hydrocephaloid symptoms in infants, if met by prompt and suitable treatment, are usually recovered from.

Treatment.—The treatment necessarily varies in the several forms of the affection, but it is in the main causal. The beneficial effect of the recumbent posture in affording immediate relief to the symptoms, and obviating permanent damage to the cerebral nutrition, must be always remembered. In acute anæmia from loss of blood, the head should be kept continuously low, stimulants freely administered, and as a penultimate resort, bandages, applied to the limbs from below upwards, may increase the proportionate supply of blood to the brain. If this fails, transfusion must be had recourse to. In chronic anæmia, sudden change of posture should be carefully avoided, and ferruginous tonics are needed. The form of iron, I believe, matters much less than is usually supposed. When the corpuscles are few, arsenic should be given with it. In severe cases, absolute physical rest is often advantageous, but this depends on the form of the anæmia. When the hæmoglobin is reduced out of proportion to the corpuscles, physical rest is of the greatest importance, greater even than fresh air. Corpuscles are oxygen-carriers in proportion to the hæmoglobin they contain. All muscular exertion uses up the oxygen, and the protoplasmic tissues suffer in their nutrition, and are incapacitated for the essential functions of digestion, circulation, &c. The beneficial effect of absolute rest in bed in these cases is often most striking. If, on the other hand, the corpuscles are rich in hæmoglobin (and in pernicious anæmia they may contain 50 per cent. more hæmoglobin than normal) gentle exercise may be permitted, because the oxygen-carriers have their full functional capacity, and the nutrition of the tissues does not suffer; if some of the oxygen is used in exercise, the supply to the tissues is not impaired as it is when every corpuscle contains far less than the normal amount of hæmoglobin.

In the cerebral anæmia of syncope, the recumbent posture, stimulants to the skin, cold water, faradisation, sinapisms, and ammonia to the nasal mucous membrane, assist the recovery of cardiac action and the return of consciousness. In all cases, carefully regulated food and stimulants are needed; beef tea should be given in small quantities at frequent intervals.

The group of symptoms called "hydrocephaloid" requires similar treatment. Its diagnosis is of the first importance, because a routine treatment for meningitis would kill the patient. Warmth to the surface of the body and head is of importance. Attempts prematurely to rouse the child to consciousness are unadvisable, but when a distinct improvement has occurred in the general state, and especially in the pulse, some mental activity probably favours the return of a normal state of the cerebral circulation.

In the more pronounced mental symptoms that result from anæmia, opiates are of service in adults, either opium by the mouth or morphia under the skin. In states of depression the dose should be small and frequent, $\frac{1}{24}$ gr. of morphia three or four times a day. In excitement a larger dose ($\frac{1}{8}$ gr.) may be given for its soporific effect, but small doses should be avoided. Small doses of nitro-glycerine may also be given in the method recommended for migraine, when cerebral symptoms are very marked.

HYPERÆMIA OF THE BRAIN.

Of all regions of cerebral pathology, that of congestion of the brain is perhaps the most obscure. We have very little precise knowledge regarding it, and, as is often the case, theory has flourished in proportion to the deficiency of fact. It was long thought that the state of the vessels of the brain after death corresponds with their condition during life, and post-mortem distension was accepted as proof that any preceding cerebral symptoms were due to congestion. Hence, an extensive symptomatology was elaborated and built up on an erroneous foundation, and it has to some extent survived its evidence. The fact was unobserved or ignored that a similar condition of the brain is equally common when there are no cerebral symptoms during life, and depends chiefly on the mode of death. Moreover, congestion of organs seems to afford so satisfactory an explanation of derangement of their functions, that the temptation to assign the condition as the cause of symptoms has often been irresistible. Even statistics have been amassed, the value of which may be estimated from the fact that in one modern text-book the history of cerebral congestion has been manifestly written from cases of pure hypochondriasis. On the other hand, partly by a reaction from this, some have doubted even the possibility of the condition. The truth lies between the two extremes, but its precise position is still undetermined, and there is room for wide difference of opinion, even among those who strive to keep their minds unbiassed. It is certain, however, that the cases in which symptoms of definite character and considerable degree can be reasonably ascribed to this cause are far from frequent.

The essential pathological condition of hyperæmia of the brain is an increase in the amount of blood within its capillaries. This may occur because there is too much blood in the arteries or too much in the veins; in the former case too much blood enters the capillaries, in the latter too little leaves them; in either case they are over-distended. The two conditions differ in their causes, nature, and effects; the one is active, the other passive congestion. In the one case, the supply of arterial blood to the capillaries is excessive; in the other, it is deficient because the circulation in them is hindered. Hence, the state of passive congestion, in which the brain is under-supplied with arterial blood, approaches that of anæmia; in both anæmia and passive congestion there is anoxæmia, but in passive congestion the capillaries contain blood that possesses an undue amount of carbonic acid and other products of metabolism—an important difference from pure anæmia.

It was at one time thought that the blood in the brain could not vary in amount because the cranio-vertebral cavity is a closed space, and this opinion is still occasionally put forward.* But the mobility of the cerebro-spinal fluid (which occupies not only the intermembranous space and the ventricles, but also the lymphatic spaces around the vessels) permits the vascular distension to vary. If the cavity were hermetically closed, the variation could be only relative, not absolute. But the numerous foramina of the cranium and vertebral canal are occupied by less resistent structures, which no doubt may yield in some degree. Moreover, the large surface veins of the spinal cord, and still more the enormous plexus outside the spinal dura mater, doubtless constitute an important means of adaptation. The less blood these contain, the more cerebro-spinal fluid can be in the spinal canal, and the more blood can be in the cerebral vessels. Further, the processes of secretion and absorption of the cerebro-spinal fluid, always in constant operation, must be influenced by the degree of pressure, and may quickly vary with it. Although the conditions during life and after death are widely different, yet we may reasonably regard the enormous variations in the total amount of blood within the cranio-vertebral canal after death in different cases, and the variations in its distribution in the nerve-centres, as evidence that considerable variations may occur during life. Some are physiological; in the child, before the fontanelles are closed, and in the adult, when a piece of the skull is removed by injury, it is seen that the brain pulsates synchronously with the heart, and that its distension also changes with the respiratory movements of the thorax.

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* See Moxon's Lectures, 'Lancet,' i, 1881.

ETIOLOGY.—The causes that produce active and passive congestion are widely different, and require separate consideration. Some act on the brain alone, others on many viscera or even on the whole vascular system; some are transient, others are permanent.

Active Congestion.—(1) *Over-action of the heart,* without corresponding contraction of the arteries, is one important cause. Whatever increases the force of the heart, and at the same time interposes no obstacle to the flow of blood into the capillary system, is a cause of active distension of the cerebral vessels. Excited action is more frequently effective than hypertrophy, because the common cause of hypertrophy is an obstruction between the heart and the capillaries. It may occur, however, from the hypertrophy that is due to aortic regurgitation, when obstruction is slight or absent; in this condition brain power is often distinctly above the average. (2) A sudden contraction of the arterioles elsewhere—as, for instance, in the skin, from exposure to cold or during a rigor—may cause transient over-filling of the cerebral vessels together with those of other viscera. (3) Dilatation of the arterioles, and consequently an increase of the blood-supply, may be produced by certain toxic agents, especially by nitrite of amyl, nitro-glycerine, and alcohol. The throbbing and headache produced by nitrite of amyl seems in itself to prove the possibility of active congestion of the brain. Morphia causes first contraction and then dilatation. Some of these agents also quicken the action of the heart, and may thus further increase the blood-supply. Emotion may have the same influence, although the evidence of this is inconclusive.* In some diseases a similar mechanism gives rise to congestion. In exophthalmic goitre, for instance, vascular dilatation is associated with cardiac over-action. In other peculiar states of the nervous system, especially in young neurotic persons, singular conditions occur, sometimes periodically, in which there are symptoms of vaso-motor paralysis of the cerebral vessels, without other cause than the peculiar neuropathic disposition, and the tendency to such disturbance is sometimes distinctly inherited. (4) Active congestion occurs also as the first stage of inflammation, and similar acute symptoms are occasionally met with, especially in children, which may even cause death. Traces of congestion are found, but no evidence of actual inflammation. The immediate cause of the condition is often obscure; excessive brain-work, or exposure to cold, has been supposed to produce it in some cases. There is no justification, however, for referring all sudden cerebral symptoms in children to congestion. Convulsions, for instance, were once thought to be always due to this cause, but there is reason to believe that they are very rarely produced by it. (5) Insolation is probably a cause of active congestion, which may or may not go on to inflammation. The modern theory, which regards the so-called sun-stroke as really heat-stroke, the result of the over-heating of the body and not of the action of the sun on the head, is probably too sweeping. The delirium of acute febrile diseases was formerly attributed to congestion of the brain, but it is more probable that the cerebral disturbance is the result of the action of the toxic blood-state on the cells of the brain, and that any congestion is of secondary origin. (6) Active congestion is supposed to be a cause of transient general cerebral symptoms in adults, especially after middle life and in the male sex. It is believed to be frequent in those of a certain build, stout men with thick necks and florid faces, and to be favoured by alcoholic excess, and by the condition termed "plethora," which, perhaps, consists in an increase in the total quantity of blood in the system. The symptoms come on suddenly, and pass away in a manner that renders it difficult to ascribe them to an organic lesion, and in rare cases the absence of such lesion has been demonstrated. They are said sometimes to follow the suppression of an habitual discharge, especially hæmorrhagic, as that from piles, or the catamenia in women.

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* The pulsation of the brain, observed in persons who have lost a piece of the cranial bone, is said to be increased by both intellectual work and emotion (Mosso, 'Reale Acad. dei Lincei,' Roma, 1880).

Partial active congestion occurs in connection with tumours and other organic lesions of the brain, and also when an artery is obstructed, and the adjacent branches of the main vessel receive too much blood. It is possible that partial congestion occurs as a primary condition, especially in certain parts of the brain, in which limited inflammation is occasionally met with; but such inflammation (as of the grey matter of the ocular nerves, p. 198) is rare, and congestion must be still more rare. Acute congestion of the medulla oblongata has been supposed to be an occasional cause of sudden death in young children, but if this agency is really at work in these cases its origin is altogether unknown.

Passive congestion is always produced mechanically by some obstruction to the return of blood. It occurs in heart disease which causes over-filling of the venous system, although the head suffers less than parts that are below the level of the heart. It is produced also by pressure on the superior vena cava, or on the innominate veins, or on the veins of the neck, by tumours, &c. The obstruction to the flow of blood through the lungs occasioned by the act of coughing, playing wind instruments, and by other severe muscular efforts with closed glottis, is a frequent cause. Slight congestion is often produced by wearing tight collars, and is shown by headache which ceases when the pressure is relieved. In death by suffocation such congestion occurs in very intense degree. The horizontal posture probably suffices to produce some degree of congestion, the venous blood losing the aid that gravitation gives to its return, and recumbency may intensify the influence of other causes. Partial passive congestion also results from pressure on, or thrombosis in, one of the cerebral veins or sinuses.

Pathological Anatomy.—In the strict sense of the word, there is scarcely any pathological anatomy of congestion of the brain. Simple active congestion disappears after death in every organ. No trace can be seen, for instance, of the congested areola around a pustule in the skin. Only the vascular distension of actual inflammation persists, lessened in degree. The lesson of general pathology is, therefore, that whenever we find after death distension of the minute vessels, without engorgement of the veins, the condition must be regarded as one of commencing inflammation, and not of mere congestion. Nor is the case otherwise with passive congestion. Intense passive congestion occurs during death from suffocation, and yet the brain may be found anæmic after death (Ackermann, Jolly). But intense venous distension occurs after death in those parts towards which gravitation draws the blood, and hence in the posterior half of the head, when this is not raised after death. The influence of gravitation may keep the blood in the vessels of congested parts, but it is never possible to distinguish the influence of the ante-mortem distension in the presence of the more powerful post-mortem effect. If mechanical congestion has occurred, not merely during the act of death, but for many hours or days previously, and gravitation keeps up the engorgement after death, the distension of the veins and capillaries is very intense; the colour of the grey substance is dark from the more abundant capillaries in it, and even the arteries may contain a good deal of blood. But if the body is so placed after death that the blood can gravitate from the head into the relaxed vessels elsewhere, no amount of simple mechanical congestion during life can be traced twelve hours after death. It is said, when long continued, to render the veins unduly tortuous, but it is doubtful whether sufficient allowance has been made for the degree of tortuosity that may be present under normal conditions, and for the influence of senile changes in the wall of the vessels. A slight degree of effusion into the ventricles probably results from passive congestion, but it is doubtful whether this is ever sufficient to cause flattening of the convolutions, and still more doubtful is the occurrence of such swelling of the whole brain as to cause such flattening, or of any general "hypertrophy" or atrophy which have been said to occur. It is probable, however, that the spaces around the vessels, which arise by a dilatation of the perivascular sheaths, are increased by mechanical congestion, although here again, apart from congestion, the variations met with are so great, and the size of these spaces is often so considerable, that the influence of congestion upon them cannot be regarded as proved. Even in young persons their size is sometimes remarkable.

Microscopical examination shows the capillaries to be unduly large in passive congestion, and it is probable that systematic measurement would show that, after this state has been long continued, their average size becomes larger than in simple post-mortem engorgement. Bulgings of the capillaries have been occasionally seen. After asphyxial modes of death it is common to find that minute vessels here and there have given way, so that the lymphatic sheath is filled with blood. Blood-pigment in the sheaths has been found in cases of slighter long-continued congestion (Bastian). Lastly, minute microscopic hæmorrhages into the cerebral substance may be found, almost constantly, in these cases.

While the pathological anatomy of congestion is thus to a considerable extent negative, it is important to remember the fact that this affords no reason for doubting the occurrence of the condition.

SYMPTOMS.—The introductory remarks show how great is the difficulty of determining the symptoms of cerebral congestion, and how much caution is desirable in accepting the statements that have been made, founded for the most part on an elaborate symptomatology that was formulated by Andral.

Passive Congestion.—The symptoms produced by passive congestion are much more marked when this is occasional, than when it is constant, uniform, and gradually produced. This is only one instance of the remarkable tolerance of the brain to pressure if slowly developed. A slight degree of mechanical congestion causes dull headache, chiefly frontal, sometimes throbbing, and sometimes accompanied by vague discomfort, described by some patients as a "confused feeling," by others as a "sense of fulness." This congestive headache is often produced by coughing. I have known a patient suffering from phthisis to seek relief, not on account of the cough, but on account of headache which was solely due to the cough. When intense, it may cause flashes of light in the eyes, or noises in the ears. Slight general convulsion may even result, usually consisting only of clonic spasm, with or without loss of consciousness. An elderly man, with chronic bronchitis and emphysema, had severe paroxysms of coughing, and at the height of each, when his face was dusky, there was a brief general clonic spasm, without loss of consciousness. Occasionally the convulsion is more distinctly epileptoid in its character, or there may be loss of consciousness without convulsion. Persistent mechanical congestion may cause also somnolence, mental dulness, disturbed sleep, or even nocturnal delirium. Vague giddiness is often complained of, but there is seldom distinct vertigo. Tingling in the limbs and general muscular inactivity have been ascribed to this cause, but it is doubtful whether unilateral symptoms ever occur.

With these symptoms are commonly associated the signs of general cephalic hyperæmia, a turgid face, and congested conjunctiva, often with viscid secretion. They are increased by constipation, by stooping, by the recumbent posture with the head low, by tight clothes about the neck, by flexion of the neck, and by all muscular effort.

Active Congestion.—The symptoms of active congestion are much more variable and much less certain. Simple excitement of the heart's action, such as results from emotion, or from nitrite of amyl, will cause throbbing and pain in the head, but in persistent overaction of the heart, as in exophthalmic goitre or aortic regurgitation with hypertrophy, cerebral symptoms are slight or absent, perhaps because the derangement is gradually induced.

The cases in which definite and considerable disturbance can be referred, with most probability, to active congestion, are those in which there are recurring paroxysms of headache, delirium, and sometimes fever, preceded by throbbing of the vessels, and sometimes by reddening of the face—symptoms of transient character and uniform recurrence. Thus, in a case related by Nothnagel,* a man aged fifty-seven had suffered from such attacks at intervals of a few weeks or months since the age of fourteen. At first there was only a sense of fulness, heat, and pain in the head, always relieved by bleeding at the nose. Afterwards each attack began with beating of the heart, heat of head, spots before the eyes, and noises in the ears; these symptoms were followed by mental excitement and irritability, screams and tears, but no actual delusions. This condition would last a few hours or a few days, but was always at once cut short by venesection. In the intervals he was perfectly well.

Brief attacks of headache with delirium may be attended with fever and special heat of head, and have been regarded as a febrile form of cerebral congestion. Whatever difficulty there may be in accepting this explanation of such cases, it is at present very difficult to find any other, especially when the attacks are brief and recur. Well-marked instances are occasionally seen in children. Thus a clever, precocious girl of six had been liable, since the age of two, to attacks of the following character, recurring at intervals of three to nine months. There was severe left-sided headache, prostration, and elevation of temperature, reaching sometimes 103°, great heat of head, occasionally delirium, and often nausea, but no vomiting. Each attack commenced suddenly and lasted a few hours; then the child went to sleep and woke up perfectly well. There is some reason to think that such attacks in childhood may pass into non-febrile migraine at a later period. Whether they are to be ascribed to a primary vaso-motor disturbance or to a "nerve storm," the opinion that an active cerebral congestion takes at least a secondary part in the morbid process may reasonably be maintained and cannot be disproved. In some cases of migraine there is evidence of active hyperæmia of the head and face during part of the attack, but it is probable that this is of secondary origin, since the attacks in which it is most marked begin with pallor only. The brief attacks of coma, pyrexia, heat of head, and other cerebral symptoms, which occur during the course of general paralysis of the insane, are commonly regarded as of congestive nature, because they pass away completely.

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* 'Ziemssen's Cyclopædia,' Bd. xi, 1, 2 Aufl., p. 49.

Acute symptoms of brain disturbance in children, resembling those of meningitis—headache, delirium or somnolence, vomiting, strabismus, inequality of pupil, various contractions and paralyses, and a prominent fontanelle—are sometimes rapidly fatal without any post-mortem evidence of active inflammation. As already stated, if congestion of the brain is the cause, it is probably that which constitutes the first stage of meningitis.

The most common severe attacks that are ascribed to cerebral congestion are those which occur in the second half of life, and are attended with sudden loss of consciousness (the so-called "congestive apoplexy"), and sometimes by transient hemiplegia, passing away in the course of a few days. The congestive nature of many of these cases is more than doubtful, and few physicians would now care to insist on the laboured description and classification of the symptoms which were current twenty years ago. We know that an actual lesion may occur in many parts of the brain, a small hæmorrhage, or a spot of softening, and may cause only general cerebral symptoms, or local symptoms of indirect origin and very brief duration. We know, too, that vascular obstruction may occur and cause no lasting symptoms, because there may be sufficient anastomoses to permit of an adequate collateral circulation. It is in these cases that the facility and reassuring character of the diagnosis of "congestion of the brain" often tempts the physician to an opinion which is, if not erroneous, at least unwarranted. But when the attack is brief, passes away completely, is attended with the signs of what is termed "plethora," and, especially when it recurs more than once, still without enduring symptoms, the diagnosis of cerebral congestion is at least permissible, and it is certain that, in many cases of the kind, no lesion has been found after death to account for the symptoms. It is commonly held, and must be admitted as possible, that cerebral congestion may cause these symptoms, even when there are no signs of plethora or of hyperæmia of the external vessels of the head, and no pallor of the face, but still greater caution should be exercised in making the diagnosis in such a state. As an instance of the condition in which the diagnosis seemed justifiable, I may mention the case of a stout, full-blooded man, about fifty-five, with a large strongly acting heart and some arterial degeneration, who, during the preceding year, had had several attacks of left-sided weakness, each lasting only a few hours; the first was attended with loss of consciousness. The day before I saw him he complained of tingling in both hands, and a few hours later vomited, went to bed, and passed a stool into the bed; then he got up and got into bed again. His temperature was found to be 102°;

he did what he was told, and answered questions, but with imperfect articulation. During the next twenty-four hours he did not speak, but was restless, tossing about in bed, sweating profusely, with a fall of temperature to 100·5°. His pulse was 80, full, but not hard, face flushed, pupils rather small. He understood imperfectly what was said to him. There was no evidence of loss of power or sensation. The symptoms all passed away in the course of a few days without the slightest indication of a local cerebral lesion. In connection with the absence of signs of cephalic congestion, we should remember that the vascular state of the face and brain may not correspond.

There is much difficulty in reconciling hemiplegic symptoms with the idea of congestion as their cause. In the vaso-motor inertia of the old, the occurrence of active congestion of any part is not easy to understand, but it is even less easy to understand that one hemisphere of the brain alone should suffer. It is possible, however, that an explanation may be found in partial arterial degeneration, which may determine the greater or less disturbance of one part of the brain.

A "convulsive form" of cerebral congestion has been described, in which severe epileptiform convulsions are the only symptom. But the relation of these to cerebral congestion is very doubtful. Formerly, as already mentioned, almost all convulsions in young children were ascribed to such congestion, and the use of the hot bath is due to a survival of the opinion; but it is certain that simple convulsions from this cause are, to say the least, extremely rare, and the beneficial effect of the method of treatment may not have a simple significance.

It is doubtful whether any form of cerebral congestion is attended by recognisable changes in the circulation within the eye, unless the whole head shares the morbid state.

DIAGNOSIS.—The most important principles of diagnosis have been already incidentally mentioned. The chief points, disregard of which causes the grossest errors of diagnosis, are two: (1) Persistent focal symptoms, such as hemiplegic weakness, however slight, exclude mere congestion. (2) Cephalic sensations, whether vague or definite in character, are alone of no diagnostic value. Such sensations are especially common in hypochondriacal patients, who often suffer much from various feelings of fulness, tightness, heat, burning, and especially from a sensation of pressure on some part of the head, generally the vertex or occiput. These sensations are intensified by annoyance and by brain work, and are vastly increased by attention. They are purely nervous sensations, pseudo-neuralgic in nature, and there is not the slightest justification for attributing them to congestion of the brain. And yet such patients, if they consult many doctors, as they usually do, are sure to be told by some that their symptoms are due to congestion of the brain, or even (with a precision that is evidence only of profound ignorance or of actual charlatanry) to "congestion of the base of the brain," a condition that probably never exists but in a pathological imagination. The most important diagnostic indication is the increase of the symptoms by influences that increase the amount of blood in the head, and their diminution by their opposite influences.

Prognosis.—The danger to life in cerebral congestion is indicated only by the intensity of the symptoms. When these are very severe, and actual coma is present, there may be danger, but in most cases the symptoms pass away completely. They are, however, prone to recur, and hence the future is uncertain, and depends chiefly on the extent to which the cause can be discovered and its influence prevented.

Treatment.—In *passive congestion* the only effective treatment is the removal of the cause of the mechanical obstruction. Many causes are unfortunately for the most part beyond our control, but cough can be moderated; the heart, if necessary, strengthened by digitalis; and in all cases many intensifying influences can be lessened. Effort should be avoided; the clothes should be loose about the neck; the patient should sleep with the head well raised and the shoulders also raised, so as to avoid any flexion of the neck.

In *active congestion* a similar attention to posture, &c., is desirable, so as to obviate any hindrance to the escape of the blood from the over-filled capillaries. In other respects the treatment must be varied according to the individual condition. Its immediate object is to lessen the amount of blood in the cerebral arteries, and there are three modes in which this may be attempted: (1) by lessening the total volume of the blood; (2) by obtaining the dilatation of vessels elsewhere, and so drawing the blood from the head; (3) by causing the dilated cerebral arteries to contract. Some of the measures employed act in more than one way.

The most direct mode of lessening the volume of the blood, venesection, is applicable only to the plethoric form of congestion, in which the face is red and turgid, and the pulse is full. The relief that it affords is immediate and great. If the propriety of venesection is doubtful, leeches may be applied, and in children this method alone is permissible. The leeches should be applied over the mastoid process, because there is a communication between the cutaneous and intra-cranial vessels through the mastoid veins. Thus we may follow the indication that is given by nose-bleeding, which often gives striking relief to cerebral congestion and has a more direct influence on the cerebral circulation than any other hæmorrhage, although the induction of nose-bleeding is impracticable, on account of the difficulty of controlling the flow of blood. Section and subsequent ligature of the temporal artery has been recommended.

Purgation and diuresis both act by diminishing the volume of the blood, and the former at least acts also by increasing the amount of blood in the capacious intestinal vessels, while both alter the constitution of the blood by removing from it effete products. The degree of purgation must depend on the strength of the patient, but in all cases it is important that the bowels should be opened, freely or gently. Constipation causes in some way cephalic discomfort, headache, &c., and intensifies the symptoms of cerebral congestion. A saline purge is the most effective, with or without a preceding mercurial. The saline should not be too much diluted, and thus a maximum osmotic action may be obtained. Both the liquid evacuations, and the thirst which follows the action of the purgative, afford proof of the removal of liquid from the blood. Diuretics are also distinctly useful. A full dose of nitrous ether and spirit of juniper may be given every three or four hours. Diaphoretics are of doubtful value, unless the symptoms follow exposure to cold—a questionable cause. Except pilocarpine, which has not yet been tried in this affection, a hot-air bath is the only effectual diaphoretic, and this, by raising the temperature of the whole body, may have a prejudicial action.

Blood may be drawn to the limbs by immersing them in warm water and keeping them dependent. Even in health, faintness may be produced by the long immersion of the feet in hot water. "Junod's boot," in which the air is partially exhausted around a limb, was formerly employed with this object, but has fallen into almost entire disuse. General constriction of the limbs by a bandage, wound round from above downwards, so as to compress the surface veins but not the deeper arteries, has a similar effect.

The third object of treatment is to obtain contraction of the arteries of the brain. It is unfortunately never possible to ascertain to what degree this object is attained, and we can only adopt those measures that seem most likely to secure it. One of these is the application of cold to the head. This should be continuous, by ice or an irrigation-tube cap. A mustard plaster to the neck is also probably of service. The application of mustard plasters to the limbs is of doubtful value; it is possible that, as some think, they may cause a reflex contraction of the cerebral arteries, but their supposed "derivative" influence, in drawing blood to the surface, must be infinitesimal unless the application is made over a very large area. It is uncertain, also, how far pathological dilatation of the vessels of one part can be influenced by drugs. Ergot, for instance, probably would have less influence on the vessels that are in a morbid state than on those elsewhere, and it might thus increase the congestion. Digitalis may be beneficial, since it causes contraction of the arterioles; it is most important to maintain the circulation as steady and uniform as possible, and to reduce undue frequency of the action of the heart. For these ends perfect tranquillity of mind and body should be secured. Alcohol and morphia should be avoided, and insomnia or restlessness treated with bromide or other gentle sedatives.

CEREBRAL HÆMORRHAGE.

Hæmorrhage may occur into the substance of the brain, or into cavities within it, or into the membranes. The term "cerebral hæmorrhage" is sometimes used as a general designation for all intra-cranial extravasations; sometimes it is applied only to hæmorrhage into the substance of the brain (including the cerebellum, pons, and medulla), and is distinguished from "meningeal hæmorrhage," in which the extravasation is into the membranes.

Etiology.—Hæmorrhage is always due to the rupture of a vessel. The rupture may be the result of injury, or may occur "spontaneously," *i. e.* as a result solely of internal causes. The vessel that bursts is usually an artery, very rarely a vein. Capillaries may also rupture, but only minute extravasations result.

The causation of cerebral hæmorrhage includes three subjects: (1) the immediate pathological conditions that lead to the rupture; (2) the influences that bring about this pathological condition; and (3) those that actually burst the vessel. These may be distinguished as the pathological, remote, and exciting causes.

Pathological Causes.—Putting aside traumatic influences, the force that ruptures an artery is the pressure of the blood within it. But as long as the walls of an artery are in a healthy state, they very rarely give way, however great may be the pressure to which they are exposed. Healthy veins may give way under extreme pressure, as in death by suffocation; but arteries do so seldom, pehaps never. When an artery ruptures, it is because the wall is so changed as to be weakened; it may give way without any abnormal degree of pressure, but more readily if the pressure is increased. The two factors, weakening of wall and increase of blood-pressure, often coincide, because increased pressure leads to degeneration of the wall. The state of the wall is the more important of the two elements, and may cause rupture when the blood-pressure is normal.

When the wall of an artery is weakened, it yields before the blood-pressure and becomes bulged. By the extension it is thinned, and thus is further weakened, until it gives way. The bulging constitutes an aneurism. The large arteries at the base or on the surface of the brain are occasionally thus dilated and thus burst, but such aneurisms also constitute "tumours," and are considered at another page. The vessels that give way in the common form of cerebral hæmorrhage are of much smaller size, and are in the substance of the brain. In these arteries, also, bulging precedes bursting, and the rupture is of the wall of a minute aneurism. Such "miliary aneurisms," as they are termed, are therefore always to be found in cases of "spontaneous" cerebral hæmorrhage. Their constancy was proved by Charcot and Bouchard, who found them in each of seventy-seven consecutive cases. They are to be discovered by washing away the cerebral substance under water, and carefully picking out the tiny thread-like vessels that remain. On these, little red grains are seen, which a low magnifying power shows to be minute aneurisms (Fig. 116). Their size varies from $\frac{1}{100}$ to $\frac{1}{25}$ of an inch. As many as a hundred have been found in a single brain. They are met with almost exclusively in the second half of life, and, although once found in a patient only twenty years old, they are as rare under forty as is cerebral hæmorrhage. Their frequency after forty increases up to at least moderate old age. The order of frequency with which they are found in different parts of the brain is, according to Charcot and Bouchard, central ganglia, cortex, pons, cerebellum, centrum ovale, middle cerebellar peduncle, crus cerebri, medulla oblongata—an order which is not far from that of hæmorrhage. In the aneurisms, the muscular coat of the vessel has disappeared, the adventitia and intima are united. The change is said to commence by the production of new tissue elements in the outer coat (Charcot and Bouchard) or in the inner coat (Zenker), and is called "arteritis" (periarteritis or endarteritis), but the evidence of its inflammatory nature seems insufficient to justify the term. No doubt the effective element in the change is the loss of the contractile and elastic elements in the coat; in consequence of this it yields more and more to the pressure of the blood, any new tissue elements that are formed being extensible and inelastic.

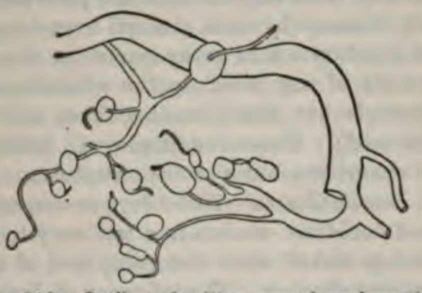

FIG. 116.—Outline of miliary aneurisms from the brain in a case of hæmorrhage; magnified. (After Eichhorst.)

The common change in the large vessels termed "atheroma" has only an indirect connection with hæmorrhage. Atheroma does not affect the small vessels within the brain, and although it renders the wall of the vessel inelastic, it also renders it thicker and less distensible than normal. Both the miliary aneurisms and atheroma are senile changes, and often co-exist, but each may be found without the other;* atheroma often exists in high degree without hæmorrhage, and the latter may occur when the vessels of the base are perfectly healthy. Nothnagel has suggested that atheroma of the larger vessels may facilitate the production of miliary aneurisms by increasing the force and suddenness of the pulse-wave that reaches the smaller arteries, the larger vessels no longer yielding to the pressure.

Miliary aneurisms are far more frequent in the brain than elsewhere, first because the external support of the cerebral arteries is slight in consequence of the wasting of the brain tissue around them; secondly, because the strain to which the vessels are exposed is greater than in other arteries of the same size, in consequence of their origin directly from large trunks, the pressure elsewhere being more gradually lessened by more gradual branching; lastly, because the absence of anastomoses in many parts (as the central ganglia) deprives the vessels of any relief to the pressure, and the pulse-wave not only distends but also elongates them, and renders them more tortuous (Mendel). These conditions will facilitate not only the production of aneurisms, but also their rupture.

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* Charcot and Bouchard found atheroma absent in one quarter of the cases of miliary aneurisms.

Remote Causes.—*Hereditary predisposition* is sometimes very marked, and doubtless consists in a tendency to the occurrence of the vascular change and of such disease as that of the kidneys, which lead to the arterial changes. The influence of heredity is shown not only by the frequency of the occurrence of cerebral hæmorrhage in some families, but by the entire freedom of other families through many long-lived generations.

Sex.—Males suffer more frequently than females, although the difference is less than is commonly asserted. Gintrac's figures of 681 cases* (excluding meningeal hæmorrhage) yield a percentage of males 56·6, females 43·4.

Age.—Cerebral hæmorrhage is essentially a disease of the degenerative period of life; at least four fifths of the cases occur after forty years of age, and its frequency increases as life advances. But it is met with occasionally in earlier life, even in childhood and infancy.† The most extensive statistics available on this point are those of Gintrac,‡ although these, consisting of published cases, are less satisfactory than a consecutive series would be, and it is probable that the frequency of the disease in early life is over-represented. The first column of the following table gives Gintrac's figures of the distribution of 653 cases he has collected, and in the second I have reduced these to a percentage. These two columns thus show the relative frequency in each decade of life. But since the number of persons living diminishes in each successive decade, such figures, as Sir George Burrows long ago insisted, convey an erroneous idea of the liability to the disease. The third column of the table is therefore computed by the help of Farr's Life Table, No. 3, and shows the number of cases of cerebral hæmorrhage in an equal number of individuals in each decade of life, called $1000x$. It thus shows the relative *liability* in each decade. Of the real value of x we have no means of judging. In the last column I have reduced the series to a percentage, so that the relative liability to the disease at each age may be more conveniently compared with its relative frequency. Thus it is seen that while the disease is actually less frequent between seventy and eighty than between sixty and seventy, the liability to it is greater in the later than in the earlier decade. From the second column it appears as if the disease were only one tenth as frequent between eighty and ninety as between fifty and sixty, but the third and fourth columns show that the liability to it is the same in these two decades. It may perhaps be questioned whether the number of cases between eighty and ninety are sufficient to justify the conclusion to which they point, that the liability is much less between eighty and ninety than between seventy and eighty, *i. e.* that in the very old cerebral hæmorrhage becomes less prevalent. But I think it probable that the conclusion is correct, and that the condition of tissues which permits the attainment of extreme old age is one in which there is less tendency to the occurrence of the arterial changes that lead to cerebral hæmorrhage.

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* 'Maladies de l'appareil nerveux,' 1869, tome ii, p. 430.

† Hæmorrhages into the substance of the brain, minute and massive, have occurred in young children during the paroxysms of whooping-cough. An instance is recorded by Marshall, 'Glasgow Med. Journal,' 1885, p. 24. It is probable that the rupture is generally of a vein. Many cases of alleged cerebral hæmorrhage in early life have been cases of extravasation into the substance of a soft glioma—always probably when optic neuritis has been found soon after the onset of the symptoms of hæmorrhage.

‡ Loc. cit., table on p. 431, with the exclusion of the cases of primary meningeal and spinal hæmorrhage.

Age.	Age distribution of cases.		Age distribution of liability.	
	No. of cases in each decade.	Percentage of cases in each decade.	Liability, number of cases in $1000x$ persons living in each decade.[*]	Percentage of liability in each decade.
1—10	15	2·3	1·8	·8
11—20	20	3·1	3	1·3
21—30	38	6·0	6	2·6
31—40	71	10·8	12	5·2
41—50	97	15·0	19	8·3
51—60	129	19·8	31	13·5
61—70	159	24·4	52	22·4
71—80	112	17·2	72	32·4
81—90	12	1·8	32	14

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[*] It may be well to explain further the manner in which the figures of the third column are obtained. From Farr's Life Table, No. 3, the population maintained in each decade by 10,000 annual births was first ascertained (the decades being, of course, reckoned according to the first column, 1—10, 11—20, &c.). The figures in the third column for each decade are the numbers that bear the same relation to 1000 as the number of cases in this decade (first column) bears to the number of persons living of that age in a population in which there are annually 10,000 births. The number 1000 is arbitrary. 1 might have been taken instead, but 1000 must be much nearer the actual number than 1 would be. The uncertain number to be added to 1000 is represented by x, which may be more or less than 1. The actual number (*i. e.* the real value of x) could not be known unless there were a post-mortem examination in every person dying in a large and known population. For the sake of simplicity, the actual population at each decade has not been given, but one

A certain physical conformation is popularly associated with a liability to "apoplexy"—a short thick neck, high shoulders, florid face; but it is doubtful whether this condition has any relation to cerebral hæmorrhage; most of the subjects are thin and spare, and present almost the reverse of the characteristic "apoplectic build."

Cerebral hæmorrhage is said to be more frequent in temperate than in tropical climates, in winter than in summer, and it is probably more common in civilised than in savage races.

Thus the degenerative tendency incidental to advanced life is, in the majority of cases, the most powerful element in the production of cerebral hæmorrhage. But this influence is augmented by certain morbid states which increase this tendency. The most important of these are chronic alcoholism, gout, and renal disease, especially the common senile form, granular disease of the kidney. Charcot found kidney disease in one third of his cases of cerebral hæmorrhage, and this is probably about the true proportion.* Whatever be the nature of fibroid disease of the kidney, whether the affection is primarily local or general, the minute vessels always suffer; and they suffer likewise, although to a less extent, in other forms of kidney disease, unquestionably primary. The strain on the vessels from the hypertrophy of the heart, whether it helps to produce the degeneration or not, must increase the tendency to dilation and rupture. Bright's disease is one of the chief causes of miliary aneurisms and cerebral hæmorrhage in persons between thirty and forty. I have elsewhere described and figured a case of Bright's disease in a woman aged thirty-six, in whose retina several miliary aneurisms could be seen with the ophthalmoscope, and who died a few days later with all the symptoms of a sudden cerebral hæmorrhage.†

Aneurisms of the larger arteries are frequently due to the two other

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instance may make the principle of the table clearer. In a population in which there are 10,000 annual births, the number of persons living between 71 and 80 will be (according to the Life Table) 1547. The number of the deaths from cerebral hæmorrhage in this period (of the cases collected) is 112. Supposing that number of deaths from this cause to occur among 1547 persons living at that period, the number of cases in 1000 will be 72, and $x = 1$. But as we do not know the exact number of persons, it is termed $1000x$. In any case, the figures represent the relative liability throughout life, so far as the cases collected by Gintrac are representative. They are doubtless not exactly representative; as before stated, the early deaths are probably in undue proportion, because such cases are more likely to be published. But, with this exception, there seems no reason why they should not fairly represent the relation of the disease to age, and they have the advantage over Registrars' reports that each case is unquestionably one of hæmorrhage, since in every case collected there was a post-mortem examination.

* It has been attempted to depreciate the influence of renal disease in the production of cerebral hæmorrhage, by demonstrating the rarity of the latter in a series of renal cases. But later adult life is essential for the occurrence of the vascular consequences, and nothing is proved by a series of cases taken at all ages.

† 'Medical Ophthalmoscopy,' pl. xii, fig. 1 (case 42, p. 326, of the 2nd ed.). In albuminuric retinitis capillary ectases are very common (ibid., fig. 68); but it is rare to meet with aneurisms on the visible arteries.

chief causes of disease of the wall, syphilitic disease and embolic arteritis (see "Intra-cranial Aneurisms"), and hæmorrhage results from their rupture. It is not yet known whether these processes also cause miliary aneurisms. I have once seen extensive hæmorrhage into the brain of a boy aged eight, with inherited syphilis and vascular disease, without any visible aneurism. The hæmorrhage had apparently commenced in the right lenticular nucleus, or outside it, and had burst into the ventricles. The syphilitic disease was chiefly in the vertebral and cerebellar arteries. I do not know of any similar case in a subject of acquired syphilis, but it is certain that the small arteries within the cerebral substance are occasionally affected by syphilitic changes, and it is possible that some cases of intra-cerebral hæmorrhage in early adult life may have this origin. Aneurism results from embolism, especially when incomplete, and the plug is from a seat of inflammation of septic character. The altered wall, still exposed to the pressure of the blood, yields before it. It may perhaps also be caused by the rapid disintegration of the embolus, the fragments of which are driven on into the minute arteries. The main trunk is sometimes pervious when there is embolic softening in its area, quickly fatal.*

Lastly, in certain general diseases there is a tendency to bleeding, which may cause intra-cerebral hæmorrhage. Thus it may occur in purpura, scurvy, pernicious anæmia, and especially in leucocythæmia, in which it is a common cause of death, and the extravasations are often multiple. Its mechanism is probably an acute degeneration in the walls of the vessels. In leucocythæmia it has been thought to be due to obstruction of the vessels by accumulations of white corpuscles, but it is not easy to see how this mechanism would be effective. In these diseases we do not know whether the vessels rupture directly, or whether they first dilate into miliary aneurisms.

Exciting Causes.—The actual rupture sometimes occurs during some temporary increase in the blood-pressure from muscular effort, such as straining at stool, lifting a heavy weight, during coitus, vomiting, or violent cough (as whooping-cough), or from excited action of the heart consequent on emotion. But often the vessel gives way when the circulation is tranquil, even during sleep. The cerebral arteries are supposed to be contracted during sleep, but it should be remembered that in the recumbent posture the influence of gravitation on the return of blood from the brain is less than in the upright

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* A remarkable case is described by Charlewood Turner ('Trans. Path. Soc.,' 1892), in which extensive embolic softening in the central ganglia of one side, from obstruction of the middle cerebral, was followed, four days later, by hæmorrhage in the same part of the other hemisphere. The heart was the seat of ulcerative endocarditis. Embolism has also been the apparent cause of hæmorrhage during convalescence from scarlet fever (Déjerine), and it has even been known to result (in the optic thalamus, bursting into the ventricle) from echinococcal embolism of the posterior cerebral (Dahnhardt, 'Neur. Cent.,' 1890.

Primary ventricular hæmorrhage is met with in rare cases, one or more of the ventricles being filled with blood without any, or with only secondary, lesions of their walls. The blood usually comes from the vessels of the choroid plexuses, or of the velum interpositum, rarely from a vein in the wall of the ventricle. Probably the hæmorrhage is due in most cases to the rupture of miliary aneurisms, which have been found in the choroid plexus, and it is for the most part related to the same general conditions as intra-cerebral hæmorrhage. But it occasionally results also from severe mechanical congestion, as in attempted hanging, from convulsions, or after a severe concussion, sometimes at an interval of a few days or one or two weeks.[*] In rare cases it proceeds from a large aneurism that has perforated the ventricle, or from a vascular growth, or occurs in hæmorrhagic diathesis, as purpura or leucocythæmia. Like meningeal hæmorrhage, it is relatively more frequent in early life than is the ordinary form, occurring even in childhood and infancy, both during birth and in the early months of life. Of ninety-four cases collected by Saunders,[†] one tenth occurred in the first year of life, and one fifth were under twenty, about a quarter under thirty, and one third under forty. It is rather more frequent in males than in females at all ages except thirty to forty, when females preponderate, in consequence of the influence of child-bearing, and the mechanism of convulsions occurring during the pregnant and puerperal states.

Traumatic hæmorrhage, the result of blows and falls on the head, may occur at any age, and is independent of arterial degeneration. It is most common in the meninges, extra-dural as the result of rupture of a meningeal artery or a sinus, or subdural or subarachnoid in consequence of rupture of a vessel of the pia mater, often as part of superficial laceration of the brain. Occasionally it occurs within the cerebral substance, and may then be multiple; *e. g.* an extravasation in the pons may co-exist with one or more extravasations into the hemispheres. Traumatic ventricular hæmorrhage is rare.

Pathological Anatomy.—In the majority of cases there is only one recent hæmorrhage in the brain; occasionally there are two or more, of which one is much larger than the other and has given rise to the symptoms. In some constitutional diseases with a tendency to

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Note continued.

Age.	Cases.	Percentage.
0—10	10	6
11—20	9	5·5
21—30	19	11·5
31—40	37	22·4
41—50	21	12·3
51—60	19	11·5
61—70	26	15·7
71—80	22	13·5
81—90	2	1·2

[*] It has been suggested that the fluid within the ventricles is driven into the aqueduct and fourth ventricle with such force as to injure the lining membrane and induce disease of its veins (Bollinger, 1891).

[†] In a careful study of this form, published in the 'American Journal of Med. Science,' vol. lxxxii, 1881, pp. 85, 337.

posture, while the blood from the lower limbs returns more readily, and thus the pressure in the arteries must be increased.

In extreme mechanical congestion, such as attends all asphyxial modes of death, small extravasations are usually produced, apart from vascular disease. They do not commonly exceed a mustard seed in size, and still more minute hæmorrhages may often be found, with the microscope, scattered through the cerebral substance, and especially numerous in the pons and medulla. Often the hæmorrhage is only into the perivascular sheath, which becomes distended with blood.

Meningeal Hæmorrhage.—Blood may be extravasated (1) outside the dura mater, separating it from the bone (extra-dural hæmorrhage); (2) beneath the dura mater, into what was regarded as the sac of the arachnoid when it was thought that a parietal layer of the arachnoid lined the dura mater (subdural hæmorrhage); and (3) beneath the arachnoid, between it and the pia mater (subarachnoid hæmorrhage). The blood may come from the arteries or veins or sinuses of the dura mater, or from the vessels of the pia mater. The chief causes are as follows:—(1) Injury that causes fracture of the skull or laceration of the pia mater. Extensive hæmorrhage is usually from the meningeal arteries or sinuses. The blood may be outside or beneath the dura mater. (2) Aneurisms of the larger arteries of the base or surface. (3) The escape of blood from an intra-cerebral hæmorrhage in the ways to be presently described. (4) Meningeal hæmorrhage occurs, apart from visible aneurisms, under the same conditions (age, chronic kidney disease, &c.) as hæmorrhage elsewhere in the brain. It is also met with in some chronic diseases with hæmorrhagic tendency, as purpura, leucocythæmia, and the malarial cachexia. The mechanism of its production in these cases is not known, but is probably the same as that which causes intra-cerebral hæmorrhage in the same conditions. (5) It occurs in the form termed "hæmatoma" already described, especially in the insane; the blood probably comes from a meningeal vein. (6) During birth it may result from the compression of the skull, especially in cases in which the head is born last. The blood comes from the vessels of the pia mater or veins of the dura mater, or even from the superior longitudinal sinus. These cases are separately described. (7) It is probably sometimes excited by exertion in a heated room, without any traceable predisposing cause.

Subarachnoid hæmorrhage is equally frequent in the two sexes, but the subdural form is three times as frequent in males as in females (Gintrac). Taking all forms together, meningeal hæmorrhage is far more frequent both in youth and in the middle period of life than intra-cerebral hæmorrhage. About one tenth of the patients do not exceed twenty years of age, and nearly half the cases occur in the first forty years of life.*

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* Gintrac's collection of 165 cases (of which only twelve were traumatic) exhibits the following distribution:

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PATHOLOGICAL ANATOMY.—In the majority of cases there is only one recent hæmorrhage in the brain; occasionally there are two or more, of which one is much larger than the other and has given rise to the symptoms. In some constitutional diseases with a tendency to

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hæmorrhage the brain may contain many extravasations. In leucocythæmia more than fifty small hæmorrhages have been found. The size of the extravasation varies from that of a nut to that of the closed fist. It may even have torn up the greater part of one hemisphere, may have distended all the ventricles, and have accumulated at the base. The two hemispheres are affected with equal frequency. Of the several parts of the brain, the central ganglia are the most frequent seat of hæmorrhage; about half the total number of extravasations commence in the corpus striatum or its neighbourhood. The clot often extends into the optic thalamus, but does not often begin in it. Next in frequency is hæmorrhage into the centrum ovale, then successively the cortex, the pons, and the cerebellum. The frequency of hæmorrhage in the cerebrum is twenty times greater that in the cerebellum. Hæmorrhage into the medulla oblongata and crus cerebri is rare, although the latter may be invaded from above, and still more so is an extravasation into the corpus callosum.[*]

Within the cerebral substance the blood occupies a cavity formed by laceration of the brain-tissue; rarely, when very small, by merely separating the fibres. The blood is clotted, and reddish black in colour; fragments of brain-tissue are mingled with it. The containing cavity is often very irregular in shape; its walls are uneven, present projecting shreds of lacerated brain-substance, and are blood-stained and softened—at first by imbibition of serum, and later by inflammation. Small extravasations are sometimes seen in the neighbourhood of a larger clot. The extravasated blood exerts pressure; the convolutions are flattened; the falx is bulged to the opposite side, and the rest of the hemisphere is anæmic. The effused blood may tear its way into the lateral ventricle; it then speedily distends both lateral ventricles and the third and fourth ventricles, and escapes by the openings at the lower extremity of the fourth ventricle, central and lateral, into the subarachnoid space. Or the blood may tear its way to the surface of the convexity, infiltrate the pia mater, and pass into the subarachnoid cavity, often by a very small opening. It has been known to force its way out of the descending cornu of the lateral ventricles by the transverse fissure. The artery from which the blood has escaped can often not be detected unless it is an aneurism of some size, and even this may be difficult to discover. The miliary aneurisms can often be found, but not always that which has ruptured.

After a time the extravasated blood undergoes changes. The clot shrinks, and gradually becomes, first chocolate, then brown, and ultimately a reddish yellow; and it then contains chiefly fat-globules, pigment and other granules, and hæmatoidin crystals. The rapidity with which it undergoes this change is uncertain, and certainly varies. It is said that the distinctive blood-colour has disappeared as early as the twentieth day; but it usually persists for a much longer time.

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[*] Erb, 'Virchow's Archiv,' xcvii, 329, in a case of cerebro-spinal meningitis.

Meanwhile the walls of the cavity undergo changes. The inflammation, in rare cases excessive and purulent, is usually conservative, and leads to the formation of connective tissue. A firm wall is thus developed, the inner surface becomes smooth, and a cyst results; sometimes it is seen in thirty or forty days, but usually only in the course of some months. It is said that connective tissue may extend across its cavity, and that in rare cases, the fluid being absorbed, the cyst walls may unite and a cicatrix result, but this is much more frequently due to softening.

Traumatic hæmorrhage occurs into and from a lacerated portion of brain, and is most frequent on the surface, especially on the convexity of each convolution. It is most frequent in regions much exposed to injury, direct or by "contre-coup," as the convexity, or the temporo-sphenoidal lobe, on the under surface of the frontal lobe. Traumatic ventricular hæmorrhage is said to result occasionally from rupture of a small vein on the surface of the corpus striatum (Prescott Hewett), or, at a later period, by the process already described.

Soft tumours (especially gliomata) are sometimes the seat of hæmorrhage which has been often mistaken for a primary extravasation, but is distinguished sometimes by the seat being one in which primary hæmorrhage is rare; and especially by the presence of some adjacent gelatinous-looking substance, which has characteristic microscopic features.

Other organs are seldom healthy; they usually present (1) the changes which indicate the predisposing causes, of which disease of the kidneys and hypertrophy of the heart are the most frequent, or (2) alterations consequent on the cerebral lesion. The lungs are loaded with serum and mucus. Occasionally intense congestions, and even hæmorrhages, are found in the stomach, &c. General congestion may be due to the mode of death, but in other cases the limited seat of the congestion and its intensity render it probable that it is due to derangement of the vaso-motor nerves.

The relation of hæmorrhages in various situations to the vascular supply has been carefully traced by Duret.* Certain arteries give way more frequently than others, and this enables us to understand the position of the extravasation. The arrangement of the arteries is described at p. 60.

Corpus Striatum.—The hæmorrhages into the corpus striatum may be divided into three series—anterior, middle, and posterior. The *anterior* are situated in the head of the caudate nucleus, and are due to the rupture of the branches that come from the anterior cerebral artery. They are usually small, but often break through into the lateral ventricle. The *middle* group comprehends those produced by the rupture of the lenticular and lenticulo-striate branches of the middle cerebral, and are the most frequent of all cerebral hæmorrhages. These vessels may rupture anywhere in their course, outside the lenticular nucleus, within it, or in the caudate nucleus. The extravasations outside the nucleus are restrained externally by the grey cortex of the insula and its subjacent layer of white substance; they may tear up or displace inwards the central ganglia. Blood within the ganglia often extends upwards and outwards in the white centrum ovale, and may attain a very large size. The *posterior* hæmorrhages are due to the rupture of the lenticulo-optic arteries of the middle cerebral, which pass through the lenticular nucleus into the anterior part of the optic thalamus. The extravasation usually commences in the thalamus, or between it and the corpus striatum, and the blood is apt to escape between the two into the lateral ventricle. They often damage the posterior (sensory) part of the internal capsule. The small arteries to the inner portion of the thalamus, which pass from the posterior cerebral, or posterior communicating, sometimes give way and cause small extravasations near the surface (internal thalamic hæmorrhages), which are very prone to rupture into the ventricle. The branches of the posterior cerebral to the hinder part of the thalamus may give rise to extravasations in this situation (posterior thalamic hæmorrhage), which may either rupture into the lateral ventricle or may extend down into the crus and even into the pons.

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* 'Arch. de Physiologie,' 1874, p. 664.

Centrum Ovale.—Large hæmorrhages usually spread into the centrum ovale from the corpus striatum. The vessels in the white substance itself are small, and give rise only to small hæmorrhages, rarely larger than a walnut, and often oval, with the long axis in the direction of the fibres. Larger extravasations are sometimes found in the white substance of the occipital lobe. These are due to the rupture of branches of the calcarine division of the posterior cerebral artery.

Cortex.—Hæmorrhages limited to the cortex may occur in almost any position, but they are rare and are usually small, although they sometimes extend into the white substance, and attain in it a larger size. This occurs chiefly when the membranes have been thickened by chronic inflammation.

Crura Cerebri.—Hæmorrhages into either the corpus striatum or optic thalamus may extend down into the crus. Those which commence in the crus are usually small, and oval in form. They may descend into the pons, but do not pass up into the central ganglia. They may be situated in the inner part of the crus, or in the outer part beneath the corpora geniculata, or in the upper part beneath the corpora quadrigemina.

Pons.—Hæmorrhages are most frequent near the middle line, from the rupture of the median branches of the basilar; and the raphe usually prevents their extension to the other side. The extravasation often has a spherical shape, and may be kept from the fourth ventricle only by a thin layer of tissue (Fig. 117), which may give way, and the blood may escape into the cavity. Sometimes small hæmorrhages extend in a transverse direction, and these are due to the rupture of the small lateral branches of the median arteries. Hæmorrhages in the lateral portions of the pons, from rupture of the radicular arteries, are rare. The vessel that most frequently gives way is the branch to the root of the fifth nerve.

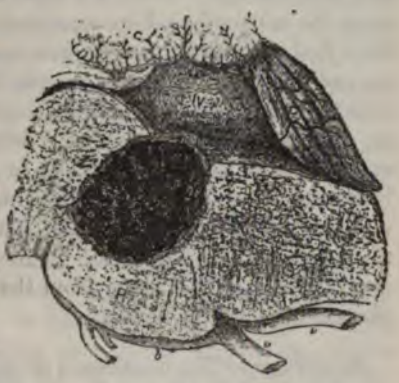

FIG. 117.—Hæmorrhage into the pons, on the left of the middle line. (After Carswell.)

Cerebellum.—Large hæmorrhages are most frequently due to the rupture of a branch that the superior cerebellar artery gives to the dentate nucleus. A small extravasation may occupy the interior of the dentate nucleus, arising from a branch of the same artery. Hæmorrhage into the inner and hinder part of the hemisphere may occur from rupture of a branch of the posterior cerebral. Extravasations near the fourth ventricle readily burst into it. The cortex of the cerebellum offers much less resistance to hæmorrhage than does the grey matter of the cerebral convolutions. The superior and inferior cerebellar peduncles are rarely the seat of hæmorrhage, but an extravasation sometimes occurs in the middle cerebellar peduncle, and, more frequently, a hæmorrhage in one half of the pons may extend a short distance into the peduncle, passing in the direction of its fibres.

In *ventricular hæmorrhage*, whether primary or secondary (by rupture of an extravasation in the substance of the brain), the blood fills all the ventricles in a third of the cases. If slowly effused, it may occupy only one lateral ventricle, and such limitation is met with in about one quarter of the cases of each variety. It occupies both lateral ventricles alone (not extending to the third and fourth), more frequently in primary than in secondary hæmorrhage. It is rarely confined to the fourth, still more rarely to the third. The blood is usually coagulated, and if small in amount the clot may correspond in form to the ventricular cavity. If the blood is slowly effused, the distension of the lateral ventricles may be less than that of the third and fourth; in the latter the accumulation of blood is aided by the influence of gravitation in the recumbent posture. In primary hæmorrhage the walls of the ventricles may be intact, the choroid plexuses are anæmic from pressure, and it may be difficult to discover the source of the blood. Superficial lacerations of the wall of the distended ventricle may be of secondary origin, as is shown by the fact that there are many of similar aspect. Sometimes it is difficult to say whether laceration is the source of the hæmorrhage or is produced by it. Occasionally there are also separate extravasations into the substance of the brain; or a meningeal hæmorrhage may co-exist, even when the ventricular extravasation is small.

The appearances in *meningeal hæmorrhage* differ according to its seat and amount. There is a layer of blood either upon the arachnoid or in the subarachnoid space, sometimes in both. The blood accumulates first in the sulci and depressions, and is generally most abundant at, and sometimes confined to, the base. If considerable in quantity over the convexity, the convolutions may be distinctly flattened. Just as, in ventricular hæmorrhage, blood may escape from the fourth ventricle into the meninges, so in extensive meningeal hæmorrhage blood may pass into the fourth ventricle, and a little may even find its way into the lateral ventricles. The blood may also pass down into the spinal canal, whether the meningeal hæmorrhage is primary, or is due to the escape of blood from the ventricles. It often distends the sheaths of the optic nerves.

SYMPTOMS.—The occurrence of cerebral hæmorrhage is indicated by symptoms of two classes, the one general and transient, the other local and more or less permanent. Both sets of symptoms are usually present, but either may be so slight as to be inconspicuous. In addition, the onset may be preceded by what are termed premonitory symptoms, and followed by symptoms outside the cerebral functions, alterations of pulse, temperature, urine, &c.

The premonitory symptoms, so called, are on the whole rare. They consist of headache, slight vertigo, weakness or tingling in the limbs, slight mental changes, or slight affection of speech. These symptoms may be continuous or paroxysmal, coming on suddenly, and passing away after a few hours or days. It is doubtful how far it is correct to speak of them as prodromata of hæmorrhage. The miliary aneurisms, however numerous, cause no symptoms until they burst. The more severe antecedent symptoms are sometimes due to the occurrence of actual small hæmorrhages, or to the commencement of a hæmorrhage that is at first slight and afterwards suddenly becomes considerable.* Slighter "prodromata" may be due to the atheroma that often co-exists with miliary aneurisms, and they are the result of interference with the blood-supply to certain parts of the brain. They thus indicate merely an associated condition. Hence they are far less frequent before attacks of hæmorrhage than before attacks of thrombotic softening, and they are similar in the two cases.

Of the general cerebral symptoms the most common is loss of consciousness; of the local symptoms, hemiplegia. The loss of consciousness usually comes on suddenly, and constitutes the most common form of "apoplexy." The patient may fall senseless, without any subjective symptom. More commonly giddiness, pain in the head, weakness in one side, or difficulty in speaking, is the first indication of the attack; and in the course of a few minutes, or longer, the patient becomes unconscious, and seems to go to sleep, but it is a sleep from which he cannot be roused, the sleep of "coma." Occasionally the obscuration of consciousness is incomplete; the patient can be partially roused, can be made to answer a question, put out his tongue, &c., but lapses again into the comatose state. In slighter cases he may only seem confused or "dull." Rarely there is not even this interference with the cerebral functions, and only the sudden onset of the local symptoms announces the occurrence of the hæmorrhage.

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* Thus a young man, who had suffered from lead-poisoning, was attacked with severe pain in the right side of the head, and after this had continued for ten days, left hemiplegia suddenly came on, without loss of consciousness. He subsequently died, and a hæmorrhage was found in the right hemisphere, which had damaged the whole thickness of the internal capsule, but had apparently occurred in two stages (Hardy, 'Gaz. méd. de Paris,' June 11, 1880).

In the most severe cases the symptoms of apoplexy, as described in a previous page, are present in the most intense degree. The muscles are relaxed and flaccid, urine and fæces escape, reflex action is abolished, not only in the limbs, but in the conjunctiva and iris. Even the knee-jerk is lost by irritative inhibition of the spinal centres. Only the beating of the heart and the movements of breathing indicate the continuance of life, while the irregularity of the former, and the laboured character and stertor of the latter, show how frail is the tenure by which life is held. In this state the patient may die a few hours after the onset, but death occurs so rapidly only when the hæmorrhage is into the pons or medulla, or distends the fourth ventricle, so as to interfere with the vital centres, cardiac or respiratory. In very rare cases death has occurred within an hour, once in as short a time as five minutes (Abercrombie). Often the coma, although complete, is less deep; the iris still acts to light, liquid placed in the mouth may still be swallowed. It is common for the muscles of one side only to be flaccid, and on the other muscular tone remains and the raised arm falls less suddenly. When the irritation is insufficient to abolish the action of the centres on which the knee-jerk, &c., depend, it may arrest the controlling influence, so that there is an early excess of this irritability, and an early foot-clonus can be obtained during the first day or two.*

The aspect of the face presents great variations. It may be flushed and turgid, or pale and pinched. The surface of the body is usually wet with perspiration. The pulse is generally at first slow, often small and incompressible, sometimes quick. Respiration, besides its labour and stertor, may have the Cheyne-Stokes rhythm, always of evil omen. The urine may be at first abundant, of low specific gravity, and acid in reaction. Occasionally albumen is present for a few hours after the onset when there is no kidney disease.

A convulsion may usher in the attack, but is not common unless the hæmorrhage is in the cortex, although hæmorrhage into the corpus striatum or elsewhere now and then causes a general convulsion. Vomiting occasionally occurs, more frequently when the hæmorrhage is into the cerebellum than when it is in other parts of the brain. Its occurrence is probably influenced in part by the condition of the stomach; if a meal has been taken shortly before the onset, the process of digestion may be arrested, and the contents of the stomach may be vomited.

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* The effect of this difference in degree is seen also after an apoplectic fit, and in the action of chloroform and ether.

The temperature usually falls within an hour after the onset, and may be only 97° or 96°; it may even reach 94·4° in the rectum.* In cases fatal during the first twelve hours, the fall may continue until death. In other cases the temperature may become normal at the end of the first day, and remain so in slight cases, or rise, in more severe forms, to two, three, or four degrees above the normal (Bourneville). But there is one apparent exception to the rule of initial depression. In cases of hæmorrhage into the pons or medulla, the temperature may rise above the normal within an hour of the onset, and during the next hour may attain a height of 104° or 106°, and the rise may go on until death. It may also rise above the normal when the hæmorrhage is into the grey matter of the central ganglia, as Hale White has shown, by experiment and observation.†

The duration of the apoplectic state varies much in cases that recover from it. At the end of from half an hour to six hours reflex action returns in the limbs, the patient gives some signs of returning consciousness, and moves those parts that are not paralysed. Difficulty of swallowing and impairment of articulation may exist for a day or two. Dull general headache is usually felt as consciousness returns, and there is occasionally some delirium.

Even during the state of apoplexy some indications of a unilateral lesion may be present; reflex action, if lost, returns on one side only; on the other it remains absent in the foot, cremaster, or abdomen: the muscular tone is different in the two arms, as already mentioned, and sometimes the affected side becomes rigid. The head and eyes may be directed to one side in "conjugate deviation," usually from the side on which the limbs are affected, sometimes towards it, according to the laws already stated (p. 78). As the general cerebral symptoms lessen, the local symptoms, persisting, become more prominent. The return of movement in the unaffected limbs defines the paralysis, which is usually hemiplegic. When the loss of consciousness is slight or absent, the loss of power is conspicuous from the first, and the subjective symptoms, already described as occasionally attending the onset of apoplexy, rise into greater prominence.

The attack often occurs during sleep. The patient is either found unconscious in the morning, or wakes unaware of what has happened, and only discovers his paralysis when he attempts to stand. The state of sleep has been thought to favour the occurrence of hæmorrhage, but it is not proved that the number of cases occurring during sleep is larger than corresponds to the proportion of existence spent in that condition.

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* In a case of extensive ventricular hæmorrhage, due to the rupture of an aneurism, recorded by Bastian ('Trans. Clin. Soc.,' 1883, p. 18).

† A gradual rise to 107·5° during the two days life endured, occurred in a case of hæmorrhage into the lenticular nucleus, observed by Pasteur ('Lancet,' 1889).

In all except the slightest cases, there occurs, two or three days after the onset, slight general febrile disturbance, due to inflammatory changes about the cerebral lesion. There may be merely headache, loss of appetite, quickening (sometimes slowing) of the pulse, and a rise of temperature of one or two degrees, lasting a few days. In other cases the rise of temperature is greater, consciousness may be dulled or even again lost, or there may be some delirium. If consciousness has not been regained, the coma deepens, there is a tendency to sloughing and vesication of the skin, swallowing is impossible, mucus accumulates in the chest, and the patient usually dies. During this stage of inflammatory reaction, rigidity ("early rigidity") often develops in the paralysed limbs. The urine becomes less abundant, and may lose its acidity.

Before the initial coma has passed away it may be suddenly deepened, or consciousness, after being regained, may be again suddenly lost, in consequence of the extravasation bursting into the ventricles. The symptoms of this occurrence are described further on.

As the general cerebral symptoms subside, only those remain that are due to the local effect of the lesion, and consist in the loss of the functions subserved by the structures destroyed. Since the hæmorrhage is usually in one side of the brain, the persisting symptoms are commonly unilateral in distribution. But the unilateral symptoms that can be at first recognised are much wider in extent than those that ultimately remain. There may be at first an apparent loss of function of almost all parts of one hemisphere, and even of the whole brain. There is not only loss of motor power, but there is often loss of sensibility, and this in cases in which the ultimate loss is purely motor. Indications of hemianopia may often be found in the early stage; if the finger is brought suddenly before the eye, first from one side and then from the other, it will be found that the eyelids blink when the finger comes from the unparalysed side, and not when it comes from the affected side. The early conjugate deviation of the head and eyes, already mentioned, is sometimes a symptom of the same character. These wide initial symptoms show that the interference with the function of the hemisphere extends at first far beyond the limits of the destruction wrought by the hæmorrhage. The effect is probably partly due to the influence of the pressure on the adjacent nerve-elements, partly to anæmia produced by that pressure, partly to inhibition by the irritative influence of the acute lesion. These symptoms gradually lessen; some, such as the hemianopia, usually pass away in a few days, others in a few weeks, until there remain only those that are the direct effect of the destruction; these persist for months, and, unless the loss is of such a character that it can be supplemented by the action of the other hemisphere, they continue for the rest of life. The pressure is, of course, greatest on the structures nearest to the hæmorrhage, and the symptoms thus produced last longer than do those that result from the slighter pressure on distant parts. The symptoms produced by these two mechanisms have been distinguished as "direct" and "indirect" symptoms.* But the two are the same in character, and can only be distinguished in practice by the gradual disappearance of the one and the persistence of the other.

In some cases there is a slight increase of the local symptoms during the period of "inflammatory reaction," due, no doubt, to the damage to adjacent structures by the inflammation around the lesion.

Chronic Stage.—The enduring symptoms, which persist after the initial stage is over, are due to the local interference with the functions of the damaged part of the brain, and are determined by the situation of the lesion. Persistent general cerebral symptoms, such, for instance, as are so conspicuous in cases of tumour, are for the most part absent in hæmorrhage. Headache is trifling; optic neuritis is practically unknown. Convulsions are rare. Some mental change may be present, slight or considerable, and evidenced chiefly by defective memory, irritability, and emotional instability, but is less than in atheromatous softening.

Local symptoms, persistent in most cases, are absent when the lesion is so placed as to spare the structures concerned directly in motion, sensation, &c.

The most common symptom is motor hemiplegia, because hæmorrhage is most frequent in the region of the corpus striatum and internal capsule, and the anterior part of the hinder line containing the fibres of the motor path rarely escapes laceration or compression. When due to damage to the motor centres or path the hemiplegia gives the characters already described, and is attended with rigidity, excess of wrist- and knee-jerk, and with the foot-clonus that indicates secondary degeneration in the pyramidal fibres. Hemianæsthesia is less common, because hæmorrhage is frequent near the posterior part of the capsule, in which the sensory tract is contained. But it is not uncommon to have some blunting of sensibility, especially on the extremities of the limbs on the hemiplegic side. Increased sensitiveness to pain is sometimes present, and may co-exist with tactile anæsthesia, and be accompanied by spontaneous pains, but these are less common than in cerebral softening. Trophic disturbances in the hemiplegic side vary in frequency and degree; there may be persistent elevation of temperature, the muscular wasting, and the joint inflammation described in the chapter on hemiplegia. The atrophy of the muscles varies in degree in different cases, but it is moderate, and is not attended with any considerable change in electrical excitability, or any destructive degeneration of the ganglion-cells of the cord.

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* A not very happy distinction, since the pressure effects are as much the direct effects of the hæmorrhage as is the laceration. Only the inhibitory symptoms are, strictly speaking, indirect, but these cannot be separated practically from those that are due to pressure.

The symptoms produced in each locality are for the most part those that have been described in the chapter on "Localisation," but a few additional facts, relating especially to hæmorrhage, may be here mentioned.

Cortex.—Hæmorrhage into the substance of the cortex is very rare, but occurs sometimes from aneurisms about which there is sufficient inflammatory thickening to prevent external rupture. This may be produced when embolism is the cause of the aneurism, and the inflammation in the wall of the artery spreads to the tissues over it. The onset is often attended by convulsions, which begin locally, and may be one-sided if the motor area is affected. Subsequent paralysis may affect only part of one side. Hæmorrhage into the centrum ovale causes symptoms similar to those due to a cortical lesion in the corresponding situation, but without symptoms of irritation of equal degree, unless the lesion is just beneath the motor cortex, when, for some reason, irritation symptoms (convulsions and rigidity) may be severe and lasting (Mills).

Crus Cerebri.—A limited extravasation may cause the characteristic crossed palsy of third nerves and limbs; but often the hæmorrhage passes beyond the limits of the crus, either upwards into the foot of the internal capsule (and then causes well-marked hemianæsthesia as well as hemiplegia) or downwards into the pons, and may then paralyse both third nerves, and the limbs on both sides. The corpora quadrigemina are never the seat of limited hæmorrhage.

Pons Varolii.—Initial loss of consciousness may be present or absent, just as in hæmorrhage elsewhere. Initial convulsions are especially frequent, rarely unilateral, but usually general; sometimes affecting the legs only, a symptom almost unknown from disease in other parts. The convulsion is often irregular in type, sometimes tonic, or tonic varied with occasional clonic jerkings. The paralysis is often bilateral, but a small hæmorrhage may cause hemiplegia from the first. Bilateral palsy may affect mainly the legs or the arms. Anæsthesia often accompanies it, and may even preponderate. The pupils are often strongly contracted, so as to suggest opium poisoning, or they may be dilated and motionless,—the difference depending on the irritation or paralysis of the nuclei of the third nerves. The deviation of the head and eyes from the side of a unilateral lesion is occasionally observed (see pp. 318 and 186). Respiration often suffers early, and may present irregularities, sometimes almost convulsive in their character. Vomiting is frequent. The temperature often rises rapidly to a hyperpyrexial degree. Death is usually more speedy than in hæmorrhage into the cerebrum; it has occurred in seven minutes.[*]

Medulla Oblongata.—A considerable hæmorrhage causes death very quickly, and even instantaneously. It is doubtful whether convulsions occur when the extravasation is confined to the medulla. The patient rarely survives the actual onset; if he does, the symptoms that persist resemble those of bulbar paralysis, but this is extremely rare; sudden apoplectiform bulbar paralysis is due, in the vast majority of cases, to softening from vascular occlusion, not to hæmorrhage.

Cerebellum.—Loss of consciousness is as common as in hæmorrhage elsewhere, and presents the same variations in its degree. There is often no initial paralysis; sometimes there is hemiplegia, due to pressure, and it may be on the same side as the hæmorrhage, or on the opposite side, according as the pressure is exerted on the pons or on the medulla. The absence of hemiplegia is more significant than its presence. The pressure may also cause various paralyses in the parts supplied by the cranial nerves that arise from the pons and medulla. The state of the pupils varies; vision is unaffected. Vomiting is more frequent than in hæmorrhage elsewhere, being met with in half the cases, and it is also more often persistent. It may occur without, as well as with, loss of consciousness. If recovery takes place, the pressure-effects, including the hemiplegia, pass away, and the only lasting symptom is cerebellar unsteadiness, which persists in some, but not in all the cases. Hæmorrhage into the cerebellum often bursts into the fourth ventricle, causing fatal depression of the functions of the medulla. When the middle cerebellar peduncle is the seat of hæmorrhage, the characteristic symptoms, forced lateral position of the head, difference in height of the eyes, and a tendency to lie on one side, and even to rotate, is usually distinct, even sometimes at the onset during the stage of apoplexy, and if the patient is conscious there is usually intense vertigo.

Ventricular Hæmorrhage.—The effusion of blood into the ventricles of the brain is indicated by severe apoplectic symptoms, the origin of which is easy to understand. When, as in the majority of cases, the rupture is into one lateral ventricle, the blood rapidly distends the other also, and the whole cortex is compressed. The blood soon passes into the third and fourth ventricles, and compresses the important structures that lie in the floor of the latter. In the rare cases in which the rupture is into the fourth ventricle the pons and medulla bear the full force of its pressure, and the grave symptoms of interference with this part of the brain are conspicuous from the first, and resemble closely those produced by hæmorrhage into the substance of the pons. This is also the case when the rupture is into the lateral ventricles, and occurs slowly; the fourth ventricle may then be much more distended than the lateral ventricles (see p. 395).

Hæmorrhage into the ventricles is sometimes primary, but much more frequently secondary to hæmorrhage into the cerebral substance, from which the blood tears its way into the cavities. In the former case the severe apoplexy is primary; in the latter it supervenes on the symptoms of the cerebral hæmorrhage already described. The primary apoplexy may have passed away or have lessened, or may be still at its height, when it suddenly returns or becomes deeper. The initial deviation of the head and eyes ceases, and is often replaced by a deviation in the opposite direction. The pulse is again slowed, and may fall to 50 or 40 beats per minute. The temperature may fall to 97° or 96°. The respiration becomes more laboured and stertorous. The reflex action is again lost in the limbs and eyes; the pupils are sometimes dilated, sometimes contracted; rigidity often appears in the limbs on the hemiplegic side, sometimes in those on the other side; there may be convulsions, sometimes general, sometimes affecting only the unparalysed side. Paroxysms of general and clonic spasm, with slight opisthotonos, have been observed.* After a few hours the pulse often becomes more frequent; the temperature may remain low or may rise, sometimes to 104° or higher. The chest becomes filled with râles, occasionally with extreme rapidity, even within a couple of hours of the onset; the face becomes livid, respiration is increasingly difficult, and the patient dies from the interference with breathing. In many cases the blood escapes from the fourth ventricle into the subarachnoid space at the base of the brain, and the symptoms may be in part due to this.

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[*] Mickle, 'British Med. Journal,' 1881, ii, 151.

Primary ventricular hæmorrhage causes symptoms which may, from the first, closely resemble those of the secondary form, but more frequently the onset resembles that of hæmorrhage into the substance of the brain, in the presence at first of unilateral symptoms. Prodromata are rare, but headache is occasionally met with, very variable in seat, character, and duration. The onset may be—(1) By sudden apoplexy, deepening rapidly; death may occur in a few hours. (2) By apoplexy with hemiplegic symptoms, or with convulsions. (3) In the very rare slow hæmorrhage, hemiplegia first occurs alone, loss of consciousness only supervening after a few hours. Hemiplegia occurs because the blood is effused first into one lateral ventricle, and causes paralysis on the opposite side by the compression of the motor path or centres. When the effusion is rapid and both lateral ventricles quickly become distended, the unilateral symptoms quickly give place to general relaxation of the muscles, and loss of all reflex action. Rigidity is often met with, but less frequently than in the secondary form; it is usually bilateral, sometimes one-sided, and occasionally involves only the muscles of mastication: it may be intermittent. Convulsions are also frequent, occurring in at least a third of the cases, sometimes general, sometimes affecting only the paralysed side, or a part of it. In cases of slow onset, speech is often lost before consciousness. The power of swallowing usually persists until the

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[*] Bastian, 'Trans. Clin. Soc.,' 1884, p. 22.

apoplexy becomes profound. The temperature resembles that of other forms of cerebral hæmorrhage. The malady is usually fatal, but recovery has occurred, shown by old and altered clot in the lateral ventricles; but it is only possible when the hæmorrhage is so small in quantity that the symptoms are slight and equivocal.

Meningeal Hæmorrhage.—The symptoms of meningeal hæmorrhage vary much according to its cause. The rupture of a large aneurism at the base causes severe apoplexy, rapidly deepening to lethal intensity. But since slight hæmorrhage sometimes occurs before the final rupture, the loss of consciousness may be preceded by definite prodromata, and is so preceded more frequently than is intra-cerebral hæmorrhage. These prodromata are severe headache (sometimes occipital), giddiness, and occasionally vomiting. The attack itself is attended by paralysis and atony of the limbs on both sides, and sometimes by palsy of cranial nerves or by convulsions.

When the hæmorrhage is of traumatic origin, the effect of the injury obscures the initial symptoms. In these and other cases in which the escape of blood is gradual, the patient may recover consciousness and continue his occupation for some hours or even for a day or two, complaining only of headache, and then gradually become somnolent and pass into a state of coma.

In some cases of meningeal hæmorrhage, convulsions are the most prominent symptom, and they may be either general or unilateral, and in the latter case may commence locally in the face or arm, or by deviation of the head. Rigidity of limb is comparatively rare, far more so than in meningitis. In some cases there is mental excitement or delirium, in others there is mental dulness. The state of the pupils is very variable; they may be contracted or dilated or unequal. Sometimes headache and giddiness are accompanied by tingling in the limbs and weakness on one or both sides. Initial apoplexy may be absent, and these symptoms, commencing suddenly, may increase until consciousness suffers. The variation presented by these cases is thus very great.

In some cases, in young adults, the occurrence of meningeal hæmorrhage is probably indicated by intense pain in the head at one spot, soon followed by coma and by convulsions. The cases are remarkable as occurring without other exciting cause than exertion in a hot room, sometimes after a meal. The patient may recover partial consciousness in a day or two, and present for about a week the symptoms of slight meningitis, but considerable mental dulness is apt to ensue, and to continue sometimes for months. Or in this, as in other forms of meningeal hæmorrhage, the patients may die in the state of coma, after a few hours; sometimes death seems due to the violence of the convulsions. Coma occasionally passes away and recurs. The frequency with which recovery occurs cannot well be estimated. The meningeal hæmorrhage of newly born children is separately described.

Pathology.—While the rupture of the vessel is always the result of weakening of its wall, and of the pressure of blood within it, the actual conditions of rupture vary considerably. The variations relate to the degree of blood-pressure—the size of the artery, of the aneurism, and of the opening in it—the freedom of exit of the blood, or the hindrance to its escape by clot within the dilated part, and the resistance in the tissue into which the blood passes, which is less in the grey than in the white substance. The precise conditions in any individual case can rarely be traced, but on them must depend the size of the hæmorrhage, and the rapidity with which the blood is effused. These two elements chiefly determine the symptoms that attend the onset. No doubt the occurrence of both aneurism and hæmorrhage is due largely to the slightness of the support that the cerebral tissue affords to its vessels. This is less in the old than in the young, on account of the larger size of the perivascular spaces, occupied only by mobile liquid.

It is a well-known law of hydrostatics that if a liquid passes into a closed chamber by a small opening, the total pressure within the chamber is that in the opening multiplied by the number of times the area of the wall of the chamber exceeds the opening in size. Thus the total pressure within the aneurism must be much greater than in the artery from which it springs, and, at the same time, the wall is weakened. After rupture, the blood in the cavity formed by the extravasation will be influenced in the same way; and although the actual condition and the result are doubtless modified, the general law must hold good in some degree. We are thus able to understand how so small a jet of blood may produce a cavity in the brain of so large a size. Doubtless the process is facilitated by the softening and disintegration of the adjacent tissue. On the other hand, the transmission of the pressure must be modified and retarded when the blood begins to coagulate.

The most common seat of hæmorrhage is the region of the corpus striatum, because the arteries that most frequently rupture are the branches that come off at right angles from the middle cerebral, in the fissure of Sylvius, and pass upwards through the lenticular nucleus and the internal capsule to the caudate nucleus and optic thalamus (see Fig. 44, p. 62). A large artery which passes between the outer part of the lenticular nucleus and the external capsule, then through the former to the internal capsule, is so frequently the source of the extravasation that it has been termed by Charcot *par excellence* "the artery of cerebral hæmorrhage." A small hæmorrhage from it simply separates the external capsule from the lenticular nucleus, occupying a narrow fissure thus formed, which may correspond to almost the whole outer aspect of the lenticular nucleus, but a large hæmorrhage displaces inwards and erodes all the central ganglia. All these branches of the middle cerebral pass to the internal capsule, and as this lies over the inner upper aspect of the lenticular nucleus, it rarely escapes damage—by pressure if the hæmorrhage is small, by laceration if it is large. Hence hemiplegia, transient or permanent, is so common a symptom. The branches that have a posterior course, to the hinder part of the capsule, less frequently give way, and hence lasting hemianæsthesia is comparatively rare. The pressure in these vessels has been found to be little less than that in the carotid (Mendel),—it is supposed because they have no anastomoses; that in the arteries of the cortex is far lower.

The mechanism by which the loss of consciousness and the other symptoms of apoplexy are produced has been previously considered (pp. 99 *et seq.*). It is only necessary here to repeat that we must recognise a double mechanism, the mechanical effect of the pressure on the cortex, and the inhibition of its cells by the mechanical irritation of the torn nerve-fibres. Both of these effects are the greater the more rapidly the blood is poured out, and the larger its amount; while the inhibitory effect is doubtless influenced also by the position of the lesion, injury to structures (as the fibres in the pons) that have an extensive connection with the cortex being specially effective.

Thus consciousness is preserved at the onset, only when the hæmorrhage is small, or the blood escapes very slowly, or when the extravasation is so placed that the irritation has but a slight influence on the cortex. When the effusion occurs slowly but the conditions are unfavorable to the cessation of the flow of blood, consciousness may be lost, not suddenly at the onset, but gradually, as the hæmorrhage attains a considerable size, giving rise to the "ingravescent apoplexy." As Broadbent has pointed out, this form is often due to rupture of the artery that passes outside the corpus striatum, although even in the case of this vessel there is sudden initial apoplexy much more frequently. The difference is probably due to the character of the rupture in the wall, whether it is large or small. Ingravescent apoplexy may also occur from rupture of a vein (as is often seen in traumatic hæmorrhage); the pressure in the vein is low, and the blood escapes slowly. It may even result from the rupture of a surface aneurism into the brain substance, the membranes being thickened and resistent.

Diagnosis.—The diagnosis of the nature of the cerebral lesion has to be made under two conditions; first during the initial apoplexy, and secondly when this has passed, and only the enduring effects of the lesion remain. The first is incomparably the most important, since by it the treatment has to be determined. Unfortunately, it is often, of all the diagnostic problems presented to the physician, at once the most difficult and the most urgent.

The first question in diagnosis is whether an attack of apoplexy is of cerebral origin. The chief points in the differential diagnosis have been considered in the account of this condition. If cerebral symptoms, such as unilateral numbness or weakness, have preceded the loss of consciousness, or are to be traced in it, as by one-sided relaxation of limb or deviation of the head, the cerebral nature of the attack is clear. If these are absent, the question must be determined by the considerations mentioned at p. 103. An actually sudden onset limits the diagnosis to the distinction from syncope, and this should be easy. If the onset was gradual, or, as is often the case, its character cannot be ascertained, the distinction has to be made from poisoning (especially by chloral, opium, and alcohol) and from uræmia, by the indications already described in the section on apoplexy.

The distinction from the apoplectiform attacks of cerebral congestion, from those of general paralysis of the insane, and from the so-called "simple apoplexy," in which there is no sign of congestion and after death no lesion of the brain, is more difficult. In all three, local symptoms are absent. In congestion the coma is rarely profound; the loss of consciousness is often imperfect, and is generally brief. The only symptoms are general; there is no local loss of power. There have usually been previous attacks of the same character, transient, leaving no after symptoms. In a first attack, or without a history, the diagnosis may be impossible. In general paralysis of the insane the preceding symptoms are almost always sufficiently pronounced to indicate the nature of the apoplectiform seizure. The attack lasts only a few hours, and the patient rapidly recovers his ordinary state. They are more readily confused with attacks of simple congestion than with hæmorrhage.

The attacks of "simple apoplexy" that occur in the old, mysterious in their nature, may resemble closely the apoplexy of cerebral hæmorrhage, and it is doubtful whether a distinction between the two is possible in practice. The so-called "serous apoplexy" is merely a variety of this form, and is important only from ignorance, and significant chiefly of the unobserved facts that are close at hand.

If the presence or history of local symptoms makes it certain, or the intensity of the general symptoms renders it in high degree probable, that the attack is due to an organic cerebral lesion, the chief diagnostic question is whether the lesion is hæmorrhage, or softening from vascular occlusion. If the patient is under forty, the presence of heart disease, or, in its absence, a suspicion of syphilis, renders vascular occlusion far more probable than hæmorrhage, provided the apoplectic symptoms are of moderate severity. But these causal indications do not absolutely exclude hæmorrhage, even in early adult life or in childhood, since both these conditions are undoubted causes of aneurism of the larger cerebral arteries. The rupture of an aneurism is probable if, under such circumstances, the apoplexy is intense in degree and the coma rapidly deepens. In a case of heart disease, preceding symptoms, headache, &c., increase the probability of aneurism.

In the second half of life, especially after forty-five, when miliary aneurisms become common, neither the presence of heart disease nor a history of syphilis affords the same strong presumption against hæmorrhage that it does in early life, and their significance is subordinate. Thrombotic softening from atheroma of the arteries becomes common *pari passu* with hæmorrhage, and it is only when the symptoms make it probable that the lesion is vascular occlusion, and not vascular rupture, that the heart disease or syphilis raises a question as to the cause of the occlusion. The older the patient the less weight do these points deserve; because atheroma increases and syphilitic disease lessens in frequency with advancing years, while the degenerative valvular disease of the old causes embolism far less frequently than do the endocarditic lesions of the young.

Between thrombotic softening and hæmorrhage the diagnosis is often difficult. The distinction is of great importance, because the treatment suitable to the two conditions is very different. A probable diagnosis can be made only by comparing the several symptoms, cerebral and general, and balancing their indications. Neither age nor sex gives help, except that in extreme old age, over eighty, there is a probability in favour of softening rather than hæmorrhage. The conformation and nutrition of the patient are not of much significance. The most important indications are those drawn from the state of the heart and circulation, the presence of conditions favouring the bursting of an artery, especially a pulse of high tension, and a strongly acting hypertrophied heart. The opposite conditions favour the formation of a clot in a degenerated vessel, and therefore thrombotic softening; so also does considerable degeneration. Irregularity of the pulse is opposed to hæmorrhage if combined with feebleness, but is unimportant if there is deep coma and the pulse is strong. Considerable degeneration of the arteries of the limbs is somewhat in favour of softening, as Nothnagel has pointed out. In Bright's disease, especially the granular kidney, there is a slight, but only a slight, probability in favour of hæmorrhage, for atheroma and softening are also frequent consequences. Hæmorrhages in the retina in connection with albuminuric retinitis do not render cerebral hæmorrhage more probable unless they are large. Visible aneurisms in the retinal arteries probably do constitute strong evidence of cerebral hæmorrhage, but they are very rare. If the attack was apparently induced immediately by strong mental excitement or physical exertion, the fact is in favour of hæmorrhage; if by prolonged grief, or the exhaustion after physical exertion, it is in favour of softening.

Prodromata of some duration in the form of numbness, tingling, or weakness in the side afterwards paralysed are in favour of atheroma and thromboses, and so also are previous headache or slight attacks of weakness in other parts. Local convulsions at the onset of the attack are also in favour of softening, because they occur chiefly in cortical disease, where softening is more common than hæmorrhage; a general convulsion suggests hæmorrhage. The significance of the occurrence of loss of consciousness at the onset depends on other associated conditions. As a general rule, loss of consciousness is more frequent, greater in degree, longer in duration, and occurs with a smaller lesion, in hæmorrhage than in softening. Its significance depends, therefore, on the probable size of the lesion. If there is reason to think that this is small, loss of consciousness is in favour of hæmorrhage; if it is probably large, the absence of apoplexy is strong evidence of softening. But it is unfortunately very difficult to judge of the extent of the lesion in the early stage. Partial hemiplegia is, however, almost always due to a small lesion, and the occurrence of initial apoplexy in such a case is in favour of hæmorrhage. Deep and prolonged coma is always in favour of hæmorrhage, unless the general indications in favour of thrombosis are very strong and the symptoms bilateral, suggesting thrombosis in both hemispheres. A considerable initial fall of temperature, exceeding 1°, or a considerable rise within a few hours, is in favour of hæmorrhage, provided the symptoms do not ndicate obstruction of the basilar, in which there may be an initial fall similar to that of hæmorrhage. Hyperpyrexia, with evidence of a lesion of the pons, may occur in either condition, but there is then seldom sudden apoplexy except in hæmorrhage. The greater the secondary inflammatory disturbance, the more probable is softening, especially if there are also secondary convulsions. In the subsequent chronic stage of hemiplegia, the most important indications are that mobile spasm (athetosis, post-hemiplegic chorea, &c.) and recurring convulsions beginning in the paralysed limb are strong evidence that the lesion was softening, and not hæmorrhage. But in this stage the history and the state of the heart and circulation also usually afford trustworthy guidance. By careful attention to these several diagnostic indications, and observing their relative weight, and comparing the direction of their significance, the cases are few in which a diagnosis of adequate probability cannot be made. It will often be found that all the indications point in one direction, and, even if not strong, derive weight from their combination, or that one important indication is decided although the others are equivocal.

The diagnosis of secondary ventricular hæmorrhage rests on the occurrence of a second apoplectic seizure, or on the distinct intensification of primary coma, with the extension to the second side of the muscular relaxation that was at first unilateral. But the significance of this depends on the initial attack having the character of hæmorrhage. When there is thrombosis in an artery of one hemisphere, the formation of another clot in a large artery on the opposite side may be attended with exactly the same symptoms as the rupture of a hæmorrhage into the ventricles; hence the occurrence of such symptoms should not be allowed to influence a diagnosis of thrombosis if the evidence of this was distinct.

The diagnosis of a primary ventricular hæmorrhage from hæmorrhage into the substance of the brain is rarely possible. There is no symptom definitely distinctive from hæmorrhage into the substance of the brain if the initial symptoms are on one side, or into the meninges if they are bilateral, unless it be, in the latter case, the suddenness and depth of coma.

In meningeal hæmorrhage the diagnostic problem differs according as there is or is not initial sudden apoplexy. If there is, the distinction is chiefly from intra-cerebral hæmorrhage, and this is often impossible from the symptoms alone. If there is no initial loss of consciousness, the symptoms are chiefly headache, delirium, and convulsions, and, developing gradually, may closely resemble those of meningitis. In all cases an important element in diagnosis is the condition under which the disease occurs. In the young, in whom alone the difficulty is likely to occur, meningeal hæmorrhage very seldom occurs except after injury. In the old, in whom spontaneous hæmorrhage may be met with, primary meningitis is very unlikely.

PROGNOSIS.—Two questions always present themselves: first, the risk of death; secondly, the prospect of recovery from the resulting paralysis. The initial danger is proportioned to the intensity and duration of the coma. If it has not begun to lessen at the end of twenty-four hours the probability is against recovery. Most cases die in which there is marked interference with respiration, indicated by its sighing character, Cheyne-Stokes rhythm, or by the accumulation of mucus in the lungs. Bilateral symptoms in the limbs are also of very grave significance, indicating, as they usually do, either hæmorrhage into the ventricles or into the pons. The lower the initial temperature the graver is the immediate prognosis. A considerable rise of temperature within a few hours of the onset is also very serious, and so is the early appearance of albumen or sugar in the urine. If the initial symptoms were severe, or the patient is old or feeble, the period of secondary inflammation is also attended with danger, the indications of which are considerable fever, delirium, or a tendency to the formation of sloughs and bedsores. Most cases die in which a slough appears on the buttock before the end of the first week. In ventricular hæmorrhage the prognosis is almost certainly fatal. In the few cases of the primary form that recover, the hæmorrhage is small in amount, and the diagnosis of the condition as a rule impossible. In meningeal hæmorrhage with coma the prognosis is exceedingly grave; but if consciousness is regained, there is a fair prospect of recovery, especially in traumatic cases, even when delirium succeeds. But there may be considerable mental impairment for weeks, or even for months.

The prognosis as regards the paralysis depends on whether it is due to direct destruction of the nerve-elements by a lesion involving the conducting path, or whether it is due to indirect damage by a lesion adjacent to it. It is usually necessary to wait for a diminution in the palsy before an opinion can be formed. The parts in which there is some return of movement before the end of a month will probably recover useful power. Paralysis that is complete at the end of three months will probably remain considerable in degree for the rest of life, except in those parts in which the other hemisphere can compensate for the loss. Thus some power always returns in the leg, except, often, in the flexors of the ankle, and it is usually at last sufficient to enable the patient to stand and walk. In the shoulder, also, there may be a return of power when the hand remains paralysed. The development of late rigidity in the hand renders recovery of power unlikely.

TREATMENT.—Little can be done to prevent the occurrence of cerebral hæmorrhage when miliary aneurisms, its main cause, are once formed. Moreover, the degenerative changes incidental to age, or the result of an inherited tendency, cannot be arrested, and perhaps not even retarded. But when the conditions likely to lead to arterial dilatation can be recognised, something may be done to prevent its occurrence by lessening its main factor, the increased strain on the wall that comes from arterial tension, and cardiac over-action, constant or occasional. A tranquil life, as free as may be from severe exertion of body or excitement of the mind, with nutritious but light food, probably has some tendency to effect these objects, and the intra-arterial pressure can be to some extent influenced by occasional saline aperients and diuretics, and by the avoidance of alcoholic excess and "high living." We cannot recognise the good we may thus effect, which is probably often considerable, if the patient's life is under our control.

In the attack itself the aim of treatment must be to secure the conditions that favour the arrest of the bleeding, *i. e.* to reduce the blood-pressure within the arteries, especially that within the cerebral vessels, and, of course, to avoid the conditions likely to increase the pressure. Physical rest is of the utmost importance. The patient should be laid down, but with the shoulders and head well raised. The clothes about the neck should be loosened, and flexion of the neck avoided, so as to prevent the hindrance to the return of blood from the head that compression of the veins would cause. All muscular effort should be forbidden. It cannot be doubted that, in many cases, the amount of hæmorrhage has been greatly increased by the patient walking about after the onset of the attack. Even passive movement should be as little as possible.

Venesection was for long regarded as the most important element in treatment; and many authorities are still of the same opinion, although the disrepute into which the lancet has fallen leads to hesitation in its use. Formerly the surgeon did not scruple, in any case of apoplexy, to cut across the temporal artery, to obtain a free flow of blood. No other agent reduces so quickly and so considerably the tension of the blood, and patients occasionally regain consciousness while the blood is flowing. On the other hand, it has been objected that the loss of blood may weaken the heart's action to a degree that would increase the peril of an extensive hæmorrhage into the brain. It is also said that, in most cases in which venesection is employed, no distinct beneficial influence can be traced. But the absence of immediate evidence of its utility is not a consideration of much weight, since the arrest of the bleeding does not, at the moment, lessen any of the conditions on which the loss of consciousness depends. But if venesection is used, it should be with discrimination. The diagnosis of hæmorrhage should be reasonably certain, since in thrombosis loss of blood will do only harm, by weakening the heart and favouring the extension of the clot. This effect, to be desired in hæmorrhage, is to be dreaded in softening. The indications for venesection are a regular, strongly acting heart, and an incompressible pulse. They are strengthened by distinct hypertrophy of the heart, by full arteries, strongly pulsating carotids, and a turgid face. The contra-indications are softness of the pulse, irregularity and dilation of the heart. If the apoplexy is so deep that the respiration and heart are suffering, it is very doubtful whether venesection can do any good. When it is indicated, the sooner the blood is drawn the better. A large opening should be made into the vein, and ten or twelve ounces allowed to flow as quickly as possible. As a means of arresting hæmorrhage from a branch of the middle cerebral, compression of the common carotid in the neck has been advocated by Spencer and Horsley,* on the ground of the observed effects, in monkeys, of a diminished flow of blood. Their observations certainly justify a trial of the method, but the diagnosis must be clear, since it would certainly favour an extension of the clot in thrombosis.

An important element is free purgation. This has a powerful influence on the cerebral circulation, by filling the capacious vessels of the intestinal canal. It should always be employed, but its slowness renders it an inefficient substitute for bleeding if the symptoms are urgent and venesection is distinctly indicated. Croton oil or calomel is the most convenient agent. With the same object a diuretic should be given.

To promote the contraction of the cerebral vessels, ice may be applied to the head, provided the patient is not collapsed, and a grain of ergotine may be injected under the skin. Mustard plasters to the nape of the neck may be employed for the same purpose, to induce reflex contraction of the arteries, if the patient's condition is one of pallor and collapse. The old practice of applying them to the soles and calves rests on an intelligible basis, since even from these distant parts an effect may be produced on the cerebral vessels, and the dilatation of the vessels of the surface must aid in attracting the blood from the brain.

Alcohol should be avoided unless the patient's collapse and cardiac depression are extreme. If a mild stimulant is indicated, ammonia may be given. It is very desirable to arrest convulsions and vomiting, because these tend to increase the bleeding. In one case, an injection of thirty grains of chloral into the rectum appeared to stop the convulsions.

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* 'Brit. Med. Journ.,' 1889, i.

In all cases, but especially if the coma is prolonged, or the secondary pyrexia considerable, the danger of the occurrence of bedsores must be borne in mind, and should be obviated as far as possible by extreme cleanliness, by a water-bed, and by changing the position of the limbs from time to time, so as to prevent long-continued pressure on the same spot. Hot-water bottles should also be employed with great care, since a degree of warmth that can be borne with impunity in health will cause a blister and slough in early hemiplegia.

In the after-treatment of these cases, when the disturbance of the onset has passed away, and slowly lessening palsy remains, it is important, with the view of preventing a recurrence, to observe the rules of life described at the commencement of this section.

The treatment of ventricular and meningeal hæmorrhage does not differ from that of the intra-cerebral form, although little can be hoped for from any measures that can be adopted.

Infantile Meningeal Hæmorrhage (Cerebral Birth Palsy).

Meningeal hæmorrhage is occasionally produced during birth; the accompanying damage to the brain may cause permanent symptoms, weakness of the limbs and of the trunk, inco-ordination, spontaneous movements, convulsions, and mental defect. The cases vary much in degree, and it is only in the more severe forms that all these symptoms are present. In some cases the legs suffer chiefly, and the condition is then termed "congenital spastic paraplegia," a malady that has been already described (vol. i, p. 441). When the spontaneous movements constitute the most conspicuous symptom, the state has been described as "congenital chorea."

The relation of these symptoms to injury to the brain during birth was suggested by Dr. Little,* and has been clearly established by the researches of an American physician, Dr. Sarah McNutt.† The following account of the symptoms is based on an extensive series of consecutive cases that have come under my notice, doubtful ones, and acquired lesions, being excluded.‡

The hæmorrhage that injures the brain is almost always due to special difficulty in parturition. In some cases (about a fifth) the presentation is unnatural, and the head is born last; in such cases intense mechanical congestion accompanies the compression, and the occurrence of hæmorrhage is easy to understand. But the same symptoms follow cases of head presentation, when the labour has been long and difficult, and its immediate effects were shown by the difficulty in getting the child to live, or by distinct symptoms of convulsions, rigidity, immobility of some part, dysphagia, &c., present from birth or coming on within a few days. About half the cases are firstborn, but in almost all the others the labour was specially difficult. In some of the cases the forceps was used, but it is probable that, as a rule, the lesion has been the result not of the use of the instruments, but of the conditions that made them necessary. In rare cases the clumsy use of the instrument may have produced the injury, as in one instance in which a permanent indentation corresponded to the centre for the opposite arm, the part chiefly affected. But it is most important that the fact should be clearly recognised that the result is not usually due to the instrumental aid, because parents are only too ready to expand even a question to an assertion, that may do great and undeserved harm to the practitioner who attended. In one case, a fifth labour, the forceps was not used, although it had been employed in each of the preceding labours, and the children had escaped injury. But I have met with a few cases in which the condition was characteristic, but the labour unusually quick and easy, and even premature. It is conceivable that the rapid birth of a skull softer than normal may cause the same intra-cranial damage as the opposite conditions. The affection seems to be almost as frequent in one sex as in the other, the proportion of girls to boys being as five to six.

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* 'Nature and Treatment of Deformities,' 1853; 'Obstetrical Trans.,' 1862.

† 'American Journal of Obstetrics,' January, 1885, and the 'Am. Journ. of Med. Science,' January, 1885. See also a valuable paper by Dr. H. R. Spencer, 'Obstet. Trans.,' vol. xxxiii, and Parrot, 'Clinique des Nouveau-nées.' These cases have also been carefully studied, especially in their later stages, and in conjunction with the acquired cerebral palsies, by Osler ('Cerebral Palsies of Children,' 1889), and by Sachs ('Journ. Nerv. and Ment. Dis.,' 1890; 'New York Med. Journ.,' 1891).

‡ Later observations fully confirm the conclusions I stated in a lecture on "Birth Palsies," 'Lancet,' 1888.

In the majority of cases nothing particular is noticed in the condition of the child during the first few days or weeks of life. In a few, however, conspicuous symptoms attract attention. There may be an evident difficulty in swallowing; one arm and leg are not moved; sometimes none of the limbs are moved, but this receives less notice than does one-sided immobility. Another frequent symptom is convulsion, general or unilateral, and often accompanied by persistent rigidity of the limbs, and with inversion of the thumbs. Rarely the head is bent back. The convulsions usually cease at the end of a week or two after birth; deglutition, if at first impaired, remains so in some degree, and there may be a conspicuous difficulty in supporting the head, which falls to one side long after the age at which infants are usually able to support it. But these symptoms are often disregarded, and very often it is not until the child is four or five months old that a deficiency of movement attracts attention; while in others, suspicion is only excited when the child fails to stand and walk at the age at which it should begin to do so. Sometimes the first thing that is noted is strong adductor spasm in the legs; in other cases it is the inability to effect ordered movements with the hands. In some instances convulsions are the most prominent symptom, and the difficulty in the use of the limbs is often ascribed to them.

The weakness, spasm, inco-ordination, and spontaneous movements usually increase during the first two years of life, being revealed by the structural and functional development of the nervous system. Their distribution varies, but all four limbs are affected in about two thirds of the cases (twenty-two out of thirty-six), and the trunk and neck muscles are also conspicuously involved in about one third (eleven cases). In a small proportion the limbs of one side only are affected (seven cases), and in rather fewer (six cases) the legs alone suffer in considerable degree. Very rarely the symptoms are confined to a single limb.

In the arms there is weakness and some spasm, which varies in character, but is greatest towards the extremity of the limb, and interferes with voluntary movement. The elbow is generally flexed, sometimes extended. The wrist is at times bent at a right angle to the forearm, at other times it is over-extended. The persistent strong flexion may even lead to subluxation. The thumbs are inverted or extended; sometimes the metacarpal bone is extended and the phalanges flexed. The fingers are flexed at the metacarpo-phalangeal joints, and in many cases they are over-extended at the middle joint, which may be subluxated. Often, however, the fingers and wrist are in constant movement, now flexed and now irregularly extended. The movement is generally slow, like the post-hemiplegic athetoid spasm (p. 86); sometimes it is quick, and may bear considerable resemblance to the movements of chorea. In slight cases the amount of spasm is trifling, and its chief effect is the interference with voluntary movement, which always exists in some degree. The fingers are separated and moved irregularly in an attempt to take hold of an object. It is often impossible for the child to pick up an object, although it can grasp firmly but slowly. In a severe case, when the hand has at last closed, it may be very difficult for the child to relax the grasp. The condition of the legs is that described and figured at p. 446, vol. i. Any attempt to move them, and any peripheral impression, excites extensor spasm, which renders the limbs rigid, the heel being drawn up and the toes pointed down. There is commonly some persistent contracture in the calf muscles, but this can almost always be completely overcome by steady pressure on the sole. Often the feet are slightly inverted, and I have once seen such extreme talipes varus that each foot was turned in and fixed at a right angle to the leg. Occasionally there is some persistent contraction of the flexors of the knee. There is often also spasm in the adductors of the thighs, which may be sufficient to carry one leg in front of the other on an attempt to stand or walk. The knee-jerk is excessive when the relaxation of the spasm permits its state to be ascertained;

a foot-clonus can seldom be obtained. Spontaneous movements of the toes and feet may occasionally be observed, and there are sometimes constantly repeated movements of flexion and extension of the trunk or limbs, especially when the mind has suffered. In very rare cases the arms suffer more than the legs. The limbs that are affected are generally thin, but there is never extreme muscular wasting. Diminished length of limb may be perceptible when the affection is one-sided, but if both sides are involved no difference can be found.

The muscles of the trunk are sometimes weakened, so that the child has a difficulty in sitting up, and, if allowed to do so, lateral curvature of the spine may be produced. There is never distinct paralysis of the face, but sometimes traces of spasm may be observed about the mouth. Occasionally difficulty in swallowing and defective articulation exist, from paralytic weakness, and sometimes associated with tremulous difficulty in moving the tongue. The defective articulation is due, in many cases, to another frequent symptom, mental defect. This exists in at least two thirds of the cases, from a doubtful amount to complete idiocy, with permanent inability to speak. In most of the cases in which mental power is low, the affection of the limbs is considerable, but the opposite rule does not hold good; the muscular affection may be severe and general, although the mind is nearly or quite normal. The difficulty in swallowing or mental defect or both, often cause dribbling of saliva, and this seems sometimes to go on as a sort of habit.

I have once seen considerable defect of sight (associated with great mental weakness); the ophthalmoscopic appearances and action of the pupils were normal. Loss of sensation in the skin is never to be found.

The affection of the two sides is seldom quite equal, and the two limbs most affected are always on the same side. Occasionally, as already stated, one side escapes, and, if the right limbs are those involved, the child is left-handed. When the legs seem to be alone affected, slight inco-ordination, or at least awkwardness of movement, may generally be observed in one arm or in both on careful examination.

Convulsive attacks occur only in a small proportion of cases, and they are rather more frequent when the affection is one-sided than when it is general. They seldom date from birth; initial convulsions rarely persist. Subsequent fits begin generally during the second half of the first year, sometimes later; they may cease after lasting for a short time, or may recur. The occurrence of fits bears no relation to the severity of the limb symptoms; and, indeed, the latter may be scarcely recognisable when the former are severe. If the limb symptoms are one-sided, so also, as a rule, are the convulsions. In a case in which the legs only were affected, and the leg centre alone found damaged, the fits began in the great toe of one side or the other.*

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* Ferguson, 'Amer. Journ. of Obstetrics,' 1891.

The subsequent course of these cases is one of slow improvement, which is greatest when age enables the will to be brought to bear on the defect; but it is only when symptoms are slight that they approximately disappear. A peculiar oscillation in walking is apt to be developed, and for a long time the patient may walk on the ball of the foot, the toes being over-extended, or upon the toes chiefly, from the talipes equinus. The knees are sometimes imperfectly extended even in walking, and there may be persistent inversion of the feet. These conditions may lessen, but often remain in some degree, so that a peculiarity of gait may persist through life.

PATHOLOGY.—In many of these cases, as we have seen, convulsions, rigidity, and paralysis are observed immediately after birth. If children die, who present such symptoms, meningeal hæmorrhage is invariably found. The extravasation is sometimes over the convexity of the brain, sometimes at the base. In the former situation it is generally bilateral, and is most considerable over the central region and towards the middle line (Fig. 118); it is found also on the medial aspect of the hemisphere (Fig. 119); this is probably due to the effects of the overlapping of the edges of the sagittal sutures. Sometimes, however, it exists only over the posterior part of the convexity. Where the layer of blood is thicker, the convolutions are much compressed, and are sometimes much injured, the cerebral tissue being broken up and infiltrated with blood. The hæmorrhage at the base is chiefly in the posterior fossa beneath the tentorium, surrounding the pons medulla and cerebellum, and it generally proceeds from a laceration in the cerebellar hemisphere. Dr. McNutt found that the basal hæmorrhage occurred in cases of head-presentation, while the most intense extravasation over the cerebral hemispheres was found in

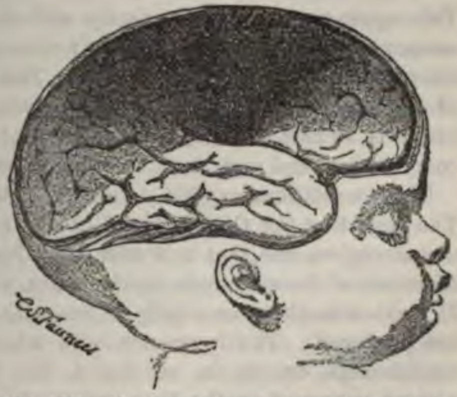

FIG. 118.—Meningeal hæmorrhage during birth. The shading represents the extravasation.

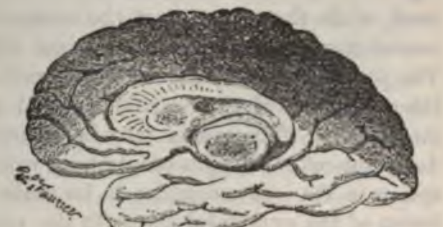

FIG. 119.—Medial aspect of the hemisphere; same case. (After McNutt.)

cases of foot-presentation. It is probable, however, that the cortical hæmorrhage also occurs in cases in which the head is born first.

In older children, who present the symptoms that have been described, the lesion found is atrophy of the convolutions in a certain region of the brain, generally the central convolutions, which may be small and indurated, and lie at the bottom of a considerable depression. This appearance agrees perfectly with the condition found in early cases, since the blood will be slowly removed, and only the damage to the cortex of the brain will remain. The long-continued compression of the convolutions hinders their development where the layer of blood is thickest, even if they are not actually damaged by the compression, and a state of atrophic sclerosis results at the spot.

FIG. 120. FIG. 121.

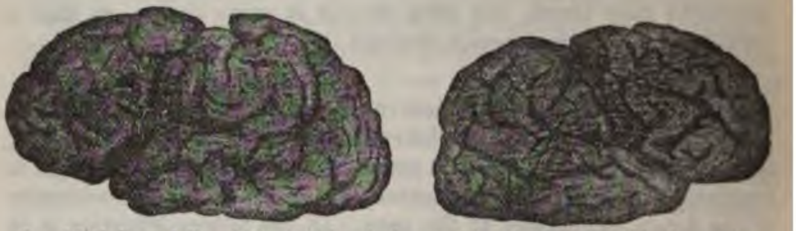

FIG. 120 left, and FIG. 121, right hemispheres of the brain of a child, the subject of bilateral birth-palsy. (After McNutt.)

These conditions explain, to a large extent, the symptoms observed. The facts that the thickest part of the extravasation is often at the motor region, and that it is over both hemispheres, explain the distribution of the symptoms in the limbs, and their bilateral character. It must sometimes be chiefly on one side, and thus unilateral effects are produced. The frequency with which the legs are involved in considerable degree is, no doubt, due to the position of the leg centres near and in the longitudinal fissure, and it is here that the extravasation is most abundant. Experiment refers to the same region the centres for the trunk muscles, which are also often weakened, while the extension of the hæmorrhage in front of the central convolutions may explain the frequent difficulty in supporting the head. The persistence of some nerve-cells only slightly damaged may explain the occurrence of the convulsions, and perhaps also the spontaneous movements. The spasm may be connected in part with the arrested development of the pyramidal fibres, which are fewer than normal. The spasm in the legs resembles, in its main features, that which follows a lesion of the pyramidal tracts in the spinal cord, and any differences are no doubt due to the occurrence of the disease during the stage of development. Whether any more complex mechanism may be concerned in producing the inco-ordination, such as the deficient control and balance of other cerebral centres, we cannot say. The extent of the damage to the cortex of the brain is a sufficient explanation of the mental defect that so often co-exists; and the fact that it is, in rare cases, chiefly over the occipital lobes, enables us to understand the impairment of sight occasionally observed.

We are not at present able to say what symptoms are due to hæmorrhage about the pons and medulla. It seems reasonable to ascribe to this the weakness of the neck and the symptoms in the region of the bulbar nerves. Nevertheless it is possible that these may be due to symmetrical lesions in the cerebral hemispheres in the lower parts of the central convolutions, and the prefrontal lobe adjacent to them, over which the hæmorrhage is apt to extend, as the figures show. It is unlikely that any limb symptoms are due to injury to the anterior pyramids of the medulla, because the symptoms are similar in all cases, and are certainly generally due to the cortical lesion. That they are the same, whether or not there is weakness of the neck, is a fact that suggests the cortical origin of the latter symptom also.

DIAGNOSIS.—In a well-marked case, in which the symptoms are general, the recognition of the disease is easy if its occurrence is known. Some difficulty is presented by cases in which the symptoms are one-sided, or in which the legs only are affected. The most important distinctions from other cerebral diseases are, first, that the condition is stationary and not progressive; and secondly, that there is no history of a distinct onset at some period subsequent to birth. Symptoms of similar character are occasionally caused by a tumour of the brain, but then gradual development and steady increase show the existence of a progressive lesion. Similar stationary symptoms may succeed an acute lesion; there is then a clear onset to be traced, often with severe symptoms, and preceded by perfect freedom from any similar affection. In the case of birth-palsy, if the derangement of movement was first noticed at a certain period, it is generally evident that attention had not been specially given to the state of the limbs. A distinct difficulty in birth, or an unnatural presentation, can generally be ascertained; and in many cases there is a history of convulsions, &c., immediately after birth, to confirm the diagnosis. When the legs suffer chiefly, the case is often thought to be disease of the spinal cord. As already mentioned, however, slight derangement of movement in the hands can generally be detected, and it should be remembered that primary chronic disease of the cord in young children is almost unknown. In any case of spastic paraplegia in a child, in whom there is no bone disease, a cerebral cause is always probable.

The greatest diagnostic difficulty is the distinction of the cases from those in which the brain has suffered before birth, either from disease or developmental defect. In such cases the birth is, as a rule, easy, although it is doubtful whether the distinction is absolute (see above). The limbs do not present the inco-ordination or rigidity, and there are no acute symptoms immediately after birth, or indication that the child has suffered during the process. On the other hand, there is usually grave mental defect, and often a history of a tendency to idiocy or nervous maladies in the family.

Prognosis.—As the damage to the brain is necessarily permanent, so are its effects. But when there is no considerable mental defect, the disorder of movement lessens in later childhood, because the will gradually acquires more power over the movement of the limbs, and is able, to some extent, to counteract the influence of the spasm. In all except the most severe cases the child ultimately acquires some ability to stand and walk, although it may be only at the age of six or eight. As already stated, the gait is often permanently peculiar. The mental condition also slowly improves. In every case, however, the prognosis must depend on the severity of the symptoms; what state the improvement will ultimately leave must depend upon the amount of defect, motor and mental, and upon the signs of improvement that can be discerned. It must be remembered also that, as a rule, the greater the defect, the later does improvement occur.

Treatment.—Drugs have no influence on the morbid state, and but little on the symptoms. Even in the early stage, soon after birth, it is doubtful whether any treatment can influence the process by which the brain is damaged. The meningeal hæmorrhage probably occurs during birth only, and there is no evidence of any subsequent increase in the extravasation. No agents can be expected to facilitate the removal of the compressing blood, which is only slowly absorbed after permanent tissue-changes have occurred in the cortex. Doubtless it could be removed by a surgical operation, but this would have to be severe, and the diagnosis would require a degree of certainty that could scarcely be obtained until changes had occurred in the brain, from which recovery would be doubtful. The slow diminution in the symptoms cannot be ascribed to the recovery of the damaged structures, since they must have passed into a stationary cicatricial condition long before improvement commences. It must be due rather to the functional education of those parts that are only slightly injured, and to the compensation effected by undamaged parts. Treatment, as far as it is possible, must consist in promoting this by training the motor power that remains, and increasing the use that it can afford, by a process of muscular education, and especially by rhythmical movements of the limbs, fingers, &c. When the inco-ordination is slight, and the child is old enough, a key type-writer is a useful means of muscular and mental education. As soon as the child becomes able to stand, the attempt to walk is, in itself, a valuable training. Instruments sometimes enable it to stand some time before this would otherwise be possible, and they may thus [illegible] accelerate improvement. It is useless, however, to employ instruments until there is enough voluntary power and control to enable the child to stand alone with their aid. Electricity in all forms is useless; its prolonged use, in cases under my care, has shown that it has no influence over either the weakness, the spasm, or the inco-ordination. The treatment of convulsive attacks is similar to that of other forms of epilepsy, but they are less readily influenced by drugs.

SOFTENING OF THE BRAIN.

Softening of the brain* is the common result of many morbid processes. The diminution of consistence is due to the breaking up of the nerve elements; the resulting particles become separated by serum, so that a soft pulp replaces the original firm tissue. It is the characteristic effect of inflammation of the brain,—and not long since all forms of softening were thought to be inflammatory. We now know that most cases of acute softening are due to a very different cause, to the arrest of the blood-supply by occlusion of an artery.† In the part supplied by the occluded vessel, the nerve elements, deprived of nutrition, at once lose their functional powers, and quickly undergo degeneration. In many parts of the brain there is not sufficient communication between the terminal arterial twigs to permit of the re-establishment of the circulation, and structural degeneration occurs in the delicate nerve elements with great rapidity; in other parts, anastomoses enable the blood-supply to be re-established, and the function returns after transient arrest. A similar result may follow occlusion of a vein: if the venous communications are insufficient to allow the blood to escape by some other channel, the circulation in the part is at an end, and the nerve elements break up; but the venous anastomoses are generally sufficient to prevent complete necrosis of the part. In rare instances there is softening that cannot be referred to either of these processes, but it is then always chronic, never acute. Thus we may distinguish, as pathological varieties of softening, Acute Softening from (1) inflammation, (2) arterial occlusion, (3) venous occlusion, and Chronic Softening. The first of these varieties will be described in the chapter on inflammation of the brain, and the last among the degenerations. Softening also results from pressure, but is a secondary and unimportant form.

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* It should be remembered that there is a considerable difference between the popular and the medical uses of the term "softening of the brain." The general meaning attached to the term is chronic mental failure, and the disease most frequently thus designated is general paralysis of the insane. In medical use the term is restricted to the condition in which the physical state actually exists. Some care must therefore be exercised in using the term, lest it convey an erroneous impression of the nature and course of the disease.

† The relation of senile softening to degeneration of the arteries was first clearly recognised by Rostan in 1823, and soon afterwards by Abercrombie, Carswell, and others. That embolism is a frequent cause of softening was first pointed out by Virchow (in the first volume of his 'Archives'), and in this country by Kirkes, who in an admirable paper ('Medico-Chir. Trans.,' 1852, p. 281) brought our knowledge of the subject within a measurable distance of its present position, and he did so apparently without being acquainted with the earlier paper of Virchow.

SOFTENING FROM ARTERIAL OCCLUSION (NECROTIC SOFTENING; ENCEPHALOMALACIA).

Softening from arterial occlusion is not only far more common than all the other forms of softening put together, but it is one of the most frequent diseases of the brain, perhaps exceeding in frequency cerebral hæmorrhage.[*]

GENERAL ETIOLOGY AND PATHOLOGY.—Two pathological processes may cause the occlusion of an artery; a plug coming from a distance may be carried into it by the blood (embolism), or a plug may be formed in it by coagulation (thrombosis). There is an important difference in the pathological relations of these two processes. Embolism is the result of a morbid process elsewhere in the circulation, commonly in the heart. Thrombosis is usually the result of local disease of an artery of the brain, by which either its calibre is narrowed, or its inner surface is changed, so as to cause coagulation; often both these conditions coincide. The chief pathological processes that cause these changes are atheroma and syphilitic disease. A change in the constitution of the blood, rendering it prone to coagulate, and slower movement of the blood, from feebleness of the heart, often aid the effect of the arterial disease in producing thrombosis, and sometimes cause it when the arteries are free from disease.

Although the primary processes of thrombosis and embolism are thus distinct, and usually occur under very different conditions, they are often conjoined in a secondary manner. If an artery is obstructed by embolism, the stagnant blood in it may clot, extending the occlusion. If a clot forms in an artery it may be detached (as Laborde has shown), and may obstruct the vessel further on by a process which is actually embolism. But the process is regarded according to its general and primary character, and the distinction between them should always be kept clearly in view.

EMBOLISM.—The source of the plug must be somewhere between the lungs and the brain,—in the pulmonary veins, the left side of the heart, the commencement of the aorta, or the large arteries of the neck. It is possible that septic material may pass through the lungs and lodge in the brain, but only capillaries can be obstructed by particles that can pass through the capillaries of the lungs, and any local softening of the brain thus produced is usually purulent, a cerebral abscess or minute points of suppuration. When an artery has been plugged in consequence of septicæmic processes in the general system (as uterine phlebitis), there has usually been either a pulmonary abscess or endocarditis to furnish the embolus. In most cases the plug comes from the heart, and its source is either diseased valves, "vegetations" from which are separated and carried along with the blood, or a coagulum in the left auricle, a fragment of the clot being detached. It is rather more common in cases of recent endocarditis, rheumatic or "ulcerative," than in chronic disease. A fresh attack of endocarditis, in valves previously diseased, is especially likely to give rise to embolism. The plug may come from either the aortic or mitral valves, but comes from the latter much more often than from the former. The most frequent cause is mitral constriction, in which several conditions probably favour the occurrence; the slow flow through the narrow orifice during diastole permits the aggregation of white corpuscles on the valve, and the quick flow during the auricular systole tends to detach the masses thus formed. Moreover, in this disease there is often great dilatation of the auricle, and clot forms in the auricular appendix; fragments of this clot are apt to be detached, or softening occurs within it, and the particles pass into the blood. The softened clot often contains organisms, and in ulcerative endocarditis, also, the particles detached (usually small) may carry infective micrococci into the cerebral arteries. Hence in both these conditions there is often plugging of minute vessels, and inflammation in the walls of the vessels and the adjacent tissue.

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[*] This is the common opinion, and is very likely correct, although the evidence is not conclusive. In the post-mortem room, hæmorrhage is the more frequent lesion, but hæmorrhage is more often quickly fatal than softening is.

Outside the heart, the plug comes, occasionally, from the lungs. Less rarely it comes from the aorta; when its wall is the seat of atheroma, fragments of degenerated tissue or of clot may be carried away by the blood. I have once known the plug to come from the interior of an aneurism. Echinococcal embolism has been once observed.

Embolism occurs with equal frequency in both sexes. It is met with at all periods of life, but is most frequent between later childhood and middle life. In the old it is less frequent, both absolutely and in comparison with thrombosis. The subjects of it have usually suffered from those diseases that are attended with endocarditis—rheumatic fever, chorea, scarlet fever, in this order of frequency. It may occur in the course of these diseases, or succeed them at any interval of time. At the time of the embolism, or soon afterwards, the signs of organic heart disease are almost always to be recognised. It occurs also, although very rarely, as a result of other acute specific diseases, any one of which is occasionally attended with endocarditis.

An immediate exciting cause is rarely to be traced. I have met with one instance in which the obstruction followed immediately a severe fright, which, by exciting the action of the heart, may have effected the detachment of the plug. Increased coagulability of the blood sometimes co-operates by causing an increase of deposit on diseased valves, or the formation of a separate clot in a feeble heart. Hence embolism often occurs a week or two after childbirth. This is a point of some practical importance: conditions favouring thrombosis do not necessarily render embolism less probable.

Thrombosis.—As we have seen, thrombosis may be due to atheroma, to syphilitic disease of the arteries, to disease of their wall by adjacent inflammation, compression, or invasion by new growth, or to a change in the blood.

Atheroma.—The larger arteries at the base of the brain are very common seats of the thickening of the inner coat, called by Virchow "Endarteritis deformans," which, when fattily degenerated, constitutes "atheroma." Opaque yellow thickenings, sometimes calcified, are the result. Only one or two of these may be present; but the change usually involves many of the larger vessels at the base, and extends for a considerable distance along the chief branches. Its distribution may be symmetrical. A similar change may exist in arteries elsewhere, or it may be confined to those of the brain. The exciting cause of this disease is probably the strain to which these arteries are exposed in consequence of their proximity to the heart and their deficient external support. It is not easy to explain their occasional freedom from atheroma when this is abundant elsewhere. The result of the degeneration is to abolish the elasticity of the vessel, to change its calibre, sometimes to lessen it or to close it altogether, sometimes to enlarge it. Alterations in the lining membrane lead to the formation of clot upon it, as on a foreign body. Where the calibre of the vessel is increased the current is retarded, and this also facilitates coagulation. The smaller arteries in the cerebral substance do not suffer in the same degree;* but the orifices of branches coming off from an atheromatous vessel are often narrowed or closed, although the main trunk is pervious, and thrombosis may be confined to these.

Atheroma is common after middle life, and increases in frequency with age. It occasionally occurs before forty, chiefly in association with chronic Bright's disease, in which the arterial tension increases the strain on the vessels. Gout also induces early degeneration. Softening from atheroma shows a corresponding relation to age. It increases in frequency as life advances, and the increase continues, if due correction is made for the diminished population, to extreme senility, exhibiting in this respect a contrast to hæmorrhage (see p. 387). The occurrence of atheroma, and therefore of softening, is facilitated by chronic alcoholism. A cachectic state of the system, or some cause (such as prostrating illness or grief) that alters the constitution of the blood, and weakens the action of the heart, often concurs in producing the thrombosis. The gouty diathesis may thus co-operate, and even cause premonitory thrombosis elsewhere.

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* Fatty degeneration of the cells lining the perivascular sheaths of the minute arteries is very common even in the young, but is without pathological significance.

Syphilitic disease is a common cause of softening.[*] It is chiefly a consequence of acquired syphilis, and so occurs in adult life. About half the sufferers are between thirty and forty at the time of the attack, one third between twenty and thirty, and most of the remainder between forty and fifty; under twenty and over fifty it is very rare. The period after infection at which it is developed varies much, but in the majority of cases is from one to twelve years. I have met with it as early as six months and as late as nineteen years after the primary sore. Fifty cases in which the diagnosis was practically certain, and the period after infection could be definitely ascertained, were distributed as follows:—In a quarter the onset was during the first two years after infection, and the remainder were distributed thus: 2—3, nine cases; 3—5, seven cases; 6—10, nine cases; 11—15, eleven cases; 16—20, four cases. The longest intervals were 18 and 19 years, the shortest 6 months (except a doubtful case at 3 months). No cases in married women are available for comparison because the date of infection cannot be ascertained; many other cases are not available because the individual has had more than one sore. The above twenty-six cases are all that could be employed for this purpose out of about a hundred of which I have notes, in which the diagnosis was reasonably certain. It has been met with in characteristic form as a result of inherited syphilis, between birth and the tenth year.

As an isolated change (apart from massive syphilitic growths) it is almost confined to the larger arteries of the base. The vessels most commonly affected are the internal carotid, middle cerebral, vertebral, basilar, and posterior cerebral. One or many vessels may be affected. The distribution of the change is commonly irregular, but sometimes it is symmetrical. The change may proceed to the obliteration of the vessel, but more frequently when the lumen is considerably narrowed a clot forms within it and suddenly arrests the circulation, or forms in branches arising from the affected part. The disease consists in a fibronuclear growth in the wall, which causes a nodular projection on the exterior (Fig. 122), and diminishes also the calibre of the vessel. The projections are more nodular, irregular, and less opaque than in atheroma. The structure of the growth resembles that of syphilomata elsewhere. It generally begins by a nuclear proliferation between the inner coat and the elastic lamina, or outside the latter: in the former case the elastic lamina is pushed outwards; it may undergo degenerative softening, and a thrombus

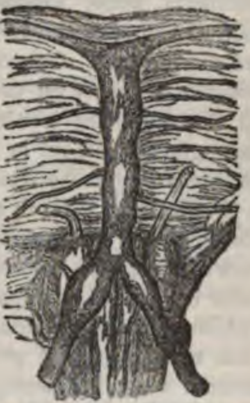

FIG. 122.—Syphilitic disease of the basilar and vertebral arteries; thrombosis of left vertebral.

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[*] See also the writer's 'Syphilis and the Nervous System,' 1893, Lect. II.

may form on the altered inner surface. Antisyphilitic treatment usually induces a fibroid cicatricial change, which causes the nodular projection to be slight, and the aspect may resemble that of early atheroma. If the elastic and muscular elements are replaced by distensible fibroid tissue, the wall may bulge into an aneurism.

Other forms of arterial disease causing thrombosis are very rare. Occasionally pressure on an artery so narrows it that coagulation occurs. Secondary arteritis may result from inflammation of the membranes, especially from traumatic meningitis. The outer coat of the vessels becomes thickened, and degenerative changes may be set up in the middle and inner coats, and may lead to thrombosis months or years after the primary mischief,* from which the patient may have apparently recovered. In the course of tubercular meningitis, a tubercular or inflammatory infiltration of the walls of the arteries has been met with, especially in small vessels, and has produced thrombosis. This occurs chiefly at the convexity, and may precede definite symptoms of inflammation, especially in adults suffering from phthisis. The inhalation of carbonic oxide has caused arterial degeneration and softening, even in young adults.† Lastly, it may be mentioned that ligature of the carotid occasionally, although rarely, causes necrotic softening of the brain. Usually the collateral circulation is sufficiently free to prevent this.

Blood-states; Simple Thrombosis.—In certain conditions of the blood, coagulation occurs with undue readiness. These states accompany childbirth, acute diseases, cancer, gout, tuberculosis, and general malnutrition at all periods of life, but especially in young children and old persons. Coinciding with disease of the arteries, it exerts a powerful influence on the occurrence of thrombosis in them. In the old this influence is often seen; the accident follows, for instance, some general illness or some depressing emotion. Arterial thrombosis may occur also from this cause when there is no arterial disease, or the clot may form in a vein or sinus. This occurs occasionally in adults, as, for instance, in the course of phthisis. It has even been known to occur in chlorosis.‡ Sudden hemiplegia sometimes occurs under conditions of impaired general health in young adults, in whom syphilis and heart disease can both be excluded; spontaneous thrombosis seems the most probable cause, since it has been observed in external veins at twenty-one, twenty-three, and twenty-four years (Angelucci). A sudden cerebral lesion frequently occurs in children, and is probably due in most cases to thrombosis in a vein or artery, but it is occasionally the result of endocarditis and embolism. These cases are separately described.

Cardiac Weakness.—The slower the blood-current, the more readily does coagulation occur. Hence feebleness of the heart's action is a powerful factor in the production of thrombosis. The weakness may be due to cardiac disease, especially to dilatation of the left ventricle, or it may be due to general causes, prostration from acute or chronic illness, and the like. Many of the general causes of thrombosis act as much or more by weakening the heart as by altering the blood.

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* 'Med. Ophthalmoscopy,' Case 4, 2nd ed., p. 270.

† Poelchen, 'Virchow's Arch.,' Bd. cxii.

‡ Skerritt, Clin. Soc., 1885.

PATHOLOGICAL ANATOMY.—*Embolism.*—The plug may consist of fibrin, usually decolourised; of vegetation, soft or firm, from a cardiac valve; sometimes of a calcareous mass from a valve of the heart or the wall of an artery; and sometimes of minute fragments resulting from the breaking down of clot or atheromatous material. In the latter case minute vessels, even capillaries, may be plugged; in the former case a large vessel is usually obstructed. Occasionally a plug, after obstructing a large vessel, breaks up, and the fragments pass on into the smaller branches. The embolus is usually arrested where the artery is narrowed by giving off a large branch, very often at a bifurcation (Fig. 123). If firm, it may retain its original shape; but if soft, it may be moulded to the form of the vessel by the pressure of the blood. A coagulum usually forms beyond the plug, and extends into the distal branches, and another often forms on the cardiac side of the plug as far as the origin of a large branch. The secondary clot is red, and from it the paler embolus can usually be distinguished without difficulty (Fig. 123, L. V.). Sometimes the distal part of the vessel is empty and contracted. The obstructing plug may break up and pass on into the minute branches before coagulation occurs. Hence we cannot always find an obstructed artery even when extensive softening has occurred.

The middle cerebral arteries and their branches are the most frequent seat of embolism, because they are the direct continuation of the carotid, and from the mode of origin of the left carotid the left middle cerebral is rather more frequently plugged than the right. But the difference between the two is less than is often stated, the proportion being as six to five.* Both middle cerebral arteries have been obstructed in rare instances.† Very seldom the internal carotid is obstructed, but the circulation in its branches is usually maintained by the circle of Willis. Next in frequency is the posterior cerebral, and then the vertebral. The left vertebral is more often plugged than the right, for the reason mentioned on p. 60; but the two posterior cerebrals are affected with equal frequency, the plug having to pass through the common basilar. The anterior cerebrals, the cerebellar arteries, and the basilar are rarely the seat of embolism. Embolism of the basilar has, indeed, been thought to be impossible,‡ but it certainly occurs, as Fig. 123 shows. A plug too large to be arrested in the vertebral, and too small to enter the posterior cerebral, must of necessity be arrested in the anterior extremity of the basilar, and the adjacent figure is an example of this.* Rarest of all is embolism of the cerebellar arteries, doubtless in consequence of the angle at which they arise from the larger trunks. More than one artery may be plugged, but usually at different times, rarely at the same time. When the plug comes from a septic source, as in ulcerative endocarditis, a secondary arteritis may occur at the obstructed part, and sometimes the inflammation may extend to the neighbouring structures. Occasionally the artery behind the plug, or when the obstruction is imperfect, is dilated into an aneurism; the walls, changed by the inflammation and rendered inelastic, yield before the pressure of the blood.

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* Gelpke, left 64 (49 per cent.), right 54 (41 per cent.)—'Arch. der Heilkunde,' 1875.

† As in a case of ulcerative endocarditis recorded by Carrington, 'Path. Trans.,' vol. xxxv, 1884, p. 108.

‡ Nothnagel, 'Topische Diagnose,' &c., 1879, p. 105; Leyden, 'Zeitsch. f. kl. Med.,' Bd. v, 1882, p. 175.

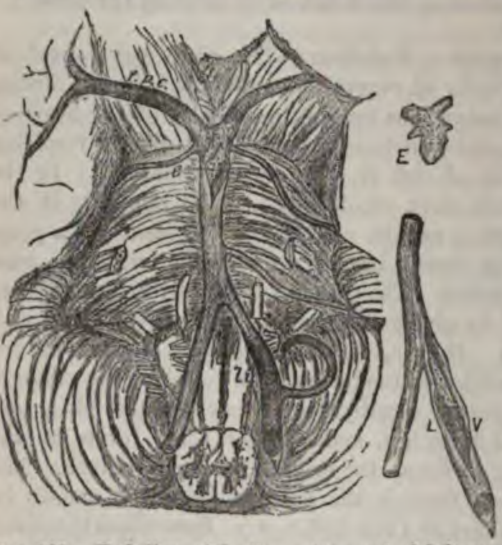

FIG. 123.—Embolism of basilar artery and of left vertebral. The latter is shown cut open at L V. The paler embolus can be distinguished from the secondary clot behind it. E is the plug *e* from the extremity of the basilar, moulded by pressure into the shape of the vessel, with projections corresponding to the branches. There is a secondary thrombus in the left post. cerebral, *l. p. c.*

Thrombosis from Atheroma.—Many arteries of the base are usually affected by atheroma, and thrombosis may occur in any of them; sometimes in two vessels simultaneously, more often in succession. It is most common in the internal carotid, middle cerebral, basilar, vertebral, and posterior cerebral. It may occur in the branches that come off from the diseased and thickened part of the vessel, although the trunk remains pervious. The branches given off by the basilar to the pons may be thus obliterated, and softening of the pons may occur In the same way the branches of the middle cerebral to the central ganglia may become closed. When a clot forms and obstructs the vessel, it usually extends into its distal branches. This is true of the internal carotid, thrombosis in which usually extends into the middle and anterior cerebrals, and even to the ophthalmic artery. In this respect thrombosis of the internal carotid presents a contrast to embolism, and has much more serious consequences. A clot in the vertebral may extend into the basilar, or the circulation in this may be maintained by the other vertebral.

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* 'Brain,' vol. i, 1882.

A clot usually obstructs the vessel where it is formed, but occasionally, as already stated, a clot that has formed on the side of a vessel may become detached, and may be arrested at a narrower part of the vessel a little farther on (Laborde). This mechanism, although probable, can rarely be proved. When the clot is in the place at which it was originally formed, it is adherent to the wall, pale, and often laminated. In a recent case the secondary coagulum that forms in the distal part of the artery is usually red and adherent. After a time it undergoes changes, may become organised, or degenerate, and even calcify. The artery may become transformed into a fibrous cord. Very rarely a thrombus breaks down, or is pushed on before distal clotting occurs, and the circulation is thus partially or entirely re-established, or the distal thrombus (especially in the cortex) only extends towards the periphery a short distance, a collateral circulation being maintained in the smaller arteries.

Syphilitic Disease.—The disease narrows the cavity of the vessel to a greater extent than does atheroma, and hence thrombosis plays a smaller but still effective part in producing the final occlusion. The branches coming off from the vessel are obstructed without occlusion of the trunk, even more frequently than in atheroma. Otherwise the two conditions produce very similar pathological effects.

Anatomical Consequences.—The first effect of arterial obstruction is to cause the arrest of the arterial blood-supply to the region supplied. There is generally arterial anæmia; sometimes the capillaries may become distended with blood from the veins, and may give way in places, so that the area may be infiltrated with hæmorrhagic points. For the first twenty-four hours there is little change in the consistence of the part, and if the vessels are empty, it is difficult to distinguish the affected area from the rest of the brain, the only difference being its paler tint. After the first twenty-four or thirty-six hours the consistence of the tissue rapidly lessens; the nerve-elements break down, and their fragments are separated by effused serum, so that an area of softening results. The serum comes no doubt partly from the blood, and partly from the lymphatic sheaths, in which the lymphatic fluid must collect if the vessels collapse. The tint of the softened area depends on the amount of blood within it, and may be white, yellow, or red from the beginning. The blood-corpuscles escape into the softening tissue, partly, perhaps, by diapedesis, and partly by the rupture of the capillaries, the walls of which undergo rapid degeneration. The red colour changes, in the course of a few weeks, to yellow, by transformation of the blood-pigment. Thus three forms of softening are distinguished—white, red, and yellow, but these do not correspond to pathological varieties. In all forms the process of destruction of the nerve-elements proceeds to the formation of fine fatty granules, in part aggregated into "granule corpuscles." These constitute a distinction from post-mortem softening, in which the segmentation of the nerve-tubes does not form such small fragments, and granule corpuscles are absent.

Red softening is found chiefly in the grey substance, where the vessels are numerous, especially in the cortex and central ganglia. The tint varies; the red colour is usually punctiform, or mingled with yellow and white. If the extravasations are large and numerous, "capillary apoplexy" results. The diminution of consistence is usually moderate. According to the amount of effusion of serum and blood, there is swelling, and the diseased area may project above the cut surface. Inflammatory changes result from the vascular distension, and in proportion to these, increase in the nuclei of the neuroglia is found. From this cause, and from the migration of white corpuscles, pus-like cells appear. The vessels are dilated, and may present a moniliform appearance. Their perivascular sheaths are often distended with blood. Commencing degeneration of the effused blood may cause a brown tint.

Yellow softening results from red softening, by degenerative changes in the blood effused. It has a similar seat, being frequently met with in the convolutions, where it constitutes the *plaques jaunes* of the French. Its consistence is usually slight, its aspect granular. The colour depends on the presence of minute pigment granules, diffused colouring matter, and hæmatoidin crystals.

White softening has the tint of the normal cerebral substance, and is chiefly found in the white substance. In consistence it varies; it may be only a little below that of the brain tissue, or it may be diffluent. Its aspect is uniform, or white flakes are scattered through it. The limits are usually gradual. Under the microscope it presents the detritus of nerve-elements, a few nuclei from the connective tissue, granule corpuscles, and, ultimately, corpora amylacea. It occasionally has a gangrenous odour, and then may be found in the white or in the grey substance, probably resulting from the obstruction of capillaries by septic material.

The process of softening is attended with inflammatory changes around the affected area, and the adjacent nerve-elements often suffer permanently in nutrition and function from this cause. It is important, in regard to the symptoms, to remember that the destroyed area is surrounded by a zone of slighter damage.

Ultimate Changes.—White and yellow softening may remain for years unchanged. Sometimes the alterations in the elements of the neuroglia and the extravasated white corpuscles result in the formation of a considerable quantity of connective tissue, consisting of fine fibre-cells and fibres, most abundant in the margins of the softened area, which become firm and dense, and trabeculæ of connective tissue may cross the cavity. After a time the fluid may be absorbed, the fat removed, and a sort of cicatrix results. In other cases the walls alone are thus altered, the solid particles are removed from the softened tissue, and a cyst is formed. The outer portion of the cyst or cicatrix may be limited by a zone of dilated blood-vessels.

Seats of Softening.—There is no part of the brain in which softening has not been found, but its most frequent seats are the cortex, the corpus striatum, and the optic thalamus. It is also frequently found in the pons, and occasionally in the medulla and cerebellum. An example of softening of the pons is shown in Fig. 59, p. 72. Its occurrence, position, and characters depend on the distribution of the vessels. The small arteries of the corpus striatum and optic thalamus are "terminal arteries," having only capillary communications with other vessels. The arteries to the surface of the brain are also sometimes terminal, but usually possess anastomoses with other branches which vary in degree and disposition. Hence obstruction in the central arteries leads invariably to softening; obstruction in the superficial arteries often causes softening, which involves the grey substance of the convolutions and some of the subjacent white centre to which the vessels penetrate; but frequently the anastomoses of the superficial vessels are so free that softening does not result, or only occurs in parts of the area of the artery occluded.* Obstruction of a main trunk (as the middle cerebral) may thus lead to softening of the central region (corpus striatum), while the convolutions escape, or both may suffer. Some parts of the cortex escape more frequently than others; the convolutions about the fissure of Sylvius are often destroyed by obstruction of the middle cerebral when the higher regions of the cortex are intact (see, for instance, the softening figured on p. 23).

The changes thus described are met with in all forms of vascular obstruction. The causes that determine the amount of blood in the softened part are not all understood, but the amount seems to be larger in the young than in the old, and perhaps in embolism than in thrombosis. Cohnheim and Litten have proved that embolism in any organ is usually attended with distension of the veins and capillaries in the infarcted area, and with abundant effusion of serum. It is surprising that such distension is not more constant in the brain.

SYMPTOMS.—The symptoms produced by the occlusion of an artery, and consequent necrotic softening of the brain, resemble those of cerebral hæmorrhage in being of two classes, one transient, the result of the sudden interference with the functions of the brain, the other more or less permanent, the result of the destruction of the nerve-elements in the affected area. The permanent symptoms depend on the position of the lesion; the symptoms that attend the onset vary according not only to the seat, but also to the nature of the vascular occlusion.

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* As an illustration of the free arterial communication that often exists in the cortex, it may be mentioned that Prof. Thane has informed me that attempts to inject separately the arteries supplying the cortex fail more often than they succeed, on account of the readiness with which the injection passes from one arterial region to another.

Premonitory symptoms are rare in embolism. When present they are not true prodromata, but consist in slighter attacks of the same character, or they are the first symptoms of the onset when the occlusion is gradual. There is no preceding indication of encephalic disease, because, until the plug obstructs the vessel, the brain is in a normal state. In thrombosis from atheroma, on the other hand, premonitory symptoms are frequent. They depend on the interference with the supply of blood, due to the disease of the vessels. They may exist for months before the onset or only for a few hours. The most common are dull general headache, giddiness, tingling, "numbness," slight weakness in one half of the body, sometimes limited to a single limb, and often, but not always, corresponding in seat to the subsequent paralysis; less commonly there is defective articulation, or some mental change, failure of memory, or irritability due to the general malnutrition of the brain, that is produced by wide-spread arterial disease. In syphilitic disease premonitory symptoms are also frequent. In some cases they resemble those just described, but more severe general cerebral symptoms are both common and characteristic. Headache is the most frequent, and is often severe, usually general, and may be worse at night. It may exist for weeks or only for a day or two before the onset. Another occasional premonitory symptom is giddiness. Sometimes there is considerable mental dulness or a somnolent condition, lasting for weeks. This is true also of other forms of arterial disease, but premonitory symptoms are absent in simple thrombosis. In all cases, their presence is far more significant than their absence.

The onset of cerebral softening is attended by loss of consciousness in a considerable number of cases, although this is less frequent than in hæmorrhage. The sudden obstruction of a large vessel, however produced, frequently entails a distinct apoplectic attack. The obstruction of a small branch, causing only a small spot of softening, often causes no loss of consciousness. For lesions of equal size, the more sudden the obstruction, the more pronounced is the apoplectic seizure. This is well seen in cases of the same nature. In embolism a gradual onset, occupying many hours, is rare, but when it occurs there is no unconsciousness; but with a sudden onset loss of consciousness is very frequent. In syphilitic disease, on the other hand, consciousness is more frequently preserved than lost. The contrast between these two conditions is more marked than between embolism and senile softening, because the advanced life renders loss of consciousness more readily produced. When there is well-marked apoplexy, the loss of consciousness may be the first symptom, but, rather more frequently than in hæmorrhage, focal symptoms, especially hemiplegia, come on before the coma. This is especially the case in atheromatous thrombosis. The apoplectic condition itself presents no characteristic feature; it resembles closely that produced by hæmorrhage. The turgid face and strongly pulsating carotids are, as a rule, absent, but these are not very common in hæmorrhage. As a rule, the degree of coma is slighter, and its duration shorter, than in hæmorrhage; but when a large vessel is plugged, the coma may be as intense and as prolonged, the resolution of the limbs as perfect, and the interference with breathing as marked, as in a severe cerebral hæmorrhage. It may last for several days, and end in death or in recovery. Coma lasting for five or six days may pass away when it is due to softening, but usually ends in death if due to hæmorrhage.

The mechanism by which apoplexy is produced by vascular obstruction can scarcely be altogether the same as in hæmorrhage, since the element of pressure is, if not absent, at least trifling in degree and late in time, due only to the œdematous swelling which comes on after the onset. Attempts have been made to explain the unconsciousness by the disturbance of the circulation. It has been ascribed, for instance, to the increase of blood-pressure in the other branches of the same vessel, owing to the pressure being suddenly diminished in the area of the infarction by the arrest of the blood-tension within it (Heubner, Wernicke). This mechanism, however, does not seem adequate. Moreover, the fact that extensive softening is sometimes unattended with apoplexy, even when quite sudden in onset, is difficult to explain, but on the whole it seems most probable that the sudden cessation of nutrition in the affected area is equivalent to a traumatic irritation, and that the loss of consciousness is chiefly due to an inhibition of the highest centres, similar to that which, in hæmorrhage, results from the laceration of the fibres.

Convulsion frequently accompanies the onset in embolism. It may be general, and irrespective of the artery plugged; sometimes it begins locally in the part afterwards most paralysed, and is then generally due to embolism of the middle cerebral, affecting the motor cortex. In atheromatous softening, and in syphilitic disease, initial convulsions are much more rare. Vomiting is not more common in any form of softening than in cerebral hæmorrhage. It is most frequent in obstruction of the basilar. Delirium may take the place of loss of consciousness in either embolism or thrombosis. In atheromatous softening, brief initial loss of consciousness is often succeeded by quiet delirium, lasting for several days or even weeks.

When there is no loss of consciousness, the onset is usually marked by sudden headache, giddiness, or incoherence, to which are quickly added the paralytic or other symptoms due to the destructive lesion. The loss of power is often accompanied by tingling in the affected side, but sometimes comes on without any associated symptom.

Focal Symptoms.—In rare instances focal symptoms are absent, the lesion being so placed as to cause none. In most cases, focal symptoms may be recognised during the period of unconsciousness, similar to those produced by hæmorrhage,—unilateral atony of muscles, loss of reflex action, inequality of face, and conjugate deviation of head and eyes. Hemiplegia is the most common form of palsy, because the middle cerebral is the artery most often plugged, and aphasia usually accompanies it when the left artery is affected. Bilateral palsy may be due to bilateral obstruction or to thrombosis of the basilar, causing softening of the pons. In the latter case, the paralysis on each side is usually partial.

The fall of temperature that attends the onset of hæmorrhage is less marked in softening, rarely exceeding half a degree, and is very often absent. A rise usually occurs in ten or twelve hours, or may be deferred for some days.* The period of inflammatory reaction is more pronounced in softening than in hæmorrhage, whatever be the cause of the softening; but it is most considerable in the old, whose brains are generally ill-nourished, and in embolism, especially when the plug comes from a septic source, such as ulcerative or septicæmic endocarditis. Any previous elevation of temperature in these cases increases considerably after the occurrence of embolism. In occlusion of the basilar artery there may be hyperpyrexia, as in hæmorrhage into the pons, but less rapidly developed, and it may be preceded by a greater depression of temperature. In the febrile disturbance, the temperature is often one or two degrees higher on the paralysed side; sloughing of the skin is common in septic embolism; and inflammation of the joints is met with less rarely than in hæmorrhage. The headache that accompanies the secondary inflammation is usually considerable, and both delirium and convulsions are not unfrequent. Optic neuritis, practically unknown from hæmorrhage, is also unknown as a result of senile (atheromatous) softening and of softening from syphilitic tumour of the arteries. If it co-exists with these it is as the result of associated causes, as Bright's disease in the former, or a syphilitic tumour in the latter case, which may co-exist with the arterial disease. But optic neuritis is met with occasionally in embolism, when the plug comes from an inflamed endocardium,† and it is apparently the consequence of the irritative character of the secondary inflammation, induced by the infective material carried by the plug. In ulcerative endocarditis, another ophthalmoscopic change may be met with—rounded hæmorrhages, with white centres, scattered over the retina. They are the result of the septic obstruction of minute vessels; similar hæmorrhages may be found in the pia mater.

Otherwise the local symptoms consist in motor and sensory paralysis, &c., according to the seat of the softening. As in hæmorrhage, the initial symptoms are more extensive than those that persist, and those that pass away during the first two weeks must be regarded as 'indirect' in origin. We cannot explain them as pressure effects, but they are no doubt the result, partly of inhibition, and partly of the interference with function from slight damage, wider than the area of actual destruction.

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* Softening of the central ganglia may be attended by a rise (Hale White). In a case of hæmorrhagic softening, outside the optic thalamus, in a girl of twenty-three, the temperature rose to 108°, but there was ulcerative endocarditis (Pasteur).

† Such cases have been recorded by Broadbent and Stephen Mackenzie, and another has come under my own observation.

The chief differences between the local symptoms in softening and hæmorrhage are the following. Aphasia is more common in softening on account of the frequency with which the cortex suffers in the region of the middle cerebral artery. Partial hemiplegia, "monoplegia," of arm, or face and arm, rarely of the leg, is also more common for the same reason; because softening affects the cortex more frequently, and such palsies are usually due to a lesion where the motor structures are separate, *i. e.* in the cortex. Another cause is that the effect of softening is more restricted than is that of hæmorrhage, on account of the absence of grave damage to adjacent parts. Recurring convulsions are also far more frequent after softening, owing to the more frequent affection of the cortex, aided by the persistence of slighter adjacent damage. In the later chronic stage, disorders of movement, choreoid, athetotic, &c., are also more common after softening than after hæmorrhage, although the explanation of the fact is still uncertain. Mental failure, loss of memory, and emotional disturbance, are rather greater in degree during the chronic stage of softening, but the difference is less than is commonly imagined, and is chiefly related to three facts: (1) foci of softening are often more numerous than those of hæmorrhage; (2) softening from atheroma is often accompanied by disease of other vessels, interfering widely with the nutrition of the brain; (3) this is also the case in syphilitic disease, in which, moreover, there may be a marked tendency to nerve degeneration.

Another point of difference is that an entire absence of focal symptoms is rather more common in softening, on account of the pressure exerted by hæmorrhage. This is often seen in lesions of the central ganglia. It is rare for a hæmorrhage limited to the caudate or lenticular nucleus not to cause hemiplegia by the pressure it exerts on the internal capsule, whereas it is not uncommon to meet with an extensive softening of one of these nuclei without any focal symptoms, past or present. Again, the onset is sometimes in two stages, especially in syphilitic disease. Slight weakness, in one case, lasted for four days before sudden hemiplegia. In another, the leg was weak one morning, and the arm and face lost power in the course of the next three or four days.

Bilateral obstruction of large vessels occurs chiefly from arterial disease; it is rarely simultaneous, but sometimes occurs in quick succession, and may then cause bilateral symptoms very closely resembling those produced by ventricular hæmorrhage. The initial disturbance may or may not have passed away when coma recurs or suddenly deepens, and quickly becomes intense with general relaxation or rigidity of muscles. The patient may die in the course of a few hours. When the vessel occluded is less important, bilateral symptoms result resembling those produced by softening of the pons. In rare cases of thrombosis or embolism the corresponding areas in the two hemispheres have been affected, cortical centres or the path from them causing symmetrical paralysis of partial extent. Thus an affection of the lower part of each motor cortex or central structures has caused not only total loss of speech, but paralysis of the lips, tongue, and throat, closely resembling that produced by a lesion of the medulla oblongata—the "pseudo-bulbar paralysis," mentioned at p. 320.

Complications.—In some forms of softening the symptoms produced by the lesion may be accompanied by others that are produced by associated morbid processes, besides those already mentioned as directly concerned in the causation of the arterial obstruction, rheumatism, gout, &c. Thus syphilitic arterial disease is sometimes associated with syphilitic meningitis, or with a syphilitic tumour. In ulcerative endocarditis the circulation of septic material in the blood often causes general fever, with rigors, headache, and delirium, enlargement and tenderness of the spleen, or hæmaturia from renal embolism. Delirium may occur without softening, in consequence of the blood-state, or of capillary obstructions in the brain or meninges; and it is not, therefore, surprising to find that it often accompanies the symptoms of an actual softening. In all cases of embolism the indications of a similar process elsewhere are frequent. An interesting complication is embolism of the central artery of the retina. It usually occurs at some other time than the cerebral embolism, but I have recorded one case in which the two accidents were simultaneous.*

Obstruction of Particular Arteries.—The distribution of the several arteries, which determines the seat of softening and the symptoms produced, has been already described (see p. 60).

Internal Carotid.—Ligature of the common carotid may cause no symptoms, or transient hemiplegia, or severe hemiplegia, ending in death at the end of a week or ten days. In the former case the communications of the circle of Willis suffice to carry on the circulation in the hemisphere. After a time the communicating arteries are found considerably dilated. Persistent hemiplegia is usually the consequence of these vessels being unusually small, so that enough blood is not conveyed to prevent necrotic softening, which may be found, post mortem, in the greater part of the hemisphere except in the region supplied by the posterior cerebral. When the communicating arteries are absent this result necessarily follows. In embolism of the internal carotid, the symptoms depend on the place at which the plug is arrested. If this is at the curve in the cavernous sinus the circulation is usually maintained in the part above by the communicating arteries, so that softening does not result, while the connection between the ophthalmic and the facial arteries helps to maintain the circulation in the former. If the plug is arrested at the division of the internal carotid, the resulting symptoms are the same as in obstruction of the middle cerebral artery; the anterior communicating conveying a supply of blood to the anterior cerebral. Thrombosis in the internal carotid consequent on atheroma may cause the same effects as embolism, but they are often more serious, because there is a greater tendency for the thrombus to spread, from the extensive disease of the wall of the vessel. Such a clot may spread into the anterior and middle cerebrals, and along the former it may pass beyond the communicating artery, producing softening of the anterior two thirds of the hemisphere, only the occipital lobe and the lower and inner portions of the temporal lobe escaping. It may spread also into the ophthalmic artery, producing (as I have seen) a necrosis of the eyeball, so that the sclerotic after death was rotten and discoloured. The symptoms of this extensive thrombosis are necessarily severe—hemiplegia with deepening coma and death at the end of (usually) four or five days. The ophthalmoscopic appearances due to the thrombosis of the ophthalmic artery are not known, but they probably resemble that produced by embolism of the central artery, perhaps with rapid opacity of the media. It is very important, in every case of apoplexy, that the eyes should be daily examined, because the obstruction of the ophthalmic artery develops gradually during coma, and subjective indications of the accident may be absent.

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* 'Medical Ophthalmoscopy,' Case 47, 2nd ed., p. 332.

The *anterior cerebral* is rarely obstructed except by plugging of the extremity of the internal carotid, because, on account of its direction at right angles to the carotid, an embolus seldom enters it. Obstruction of the first part of the vessel may cause a small spot of softening in the head of the caudate nucleus, but not elsewhere in the region supplied, unless the anterior communicating artery is absent, or the thrombus extends beyond its junction. Moreover, a collateral circulation from the branches of the middle cerebral may aid in maintaining the nutrition of the cortex. Hence softening in the cortical region supplied by this vessel is rare, except in association with obstruction of the middle cerebral. Softening of the olfactory bulb and adjacent part of the orbital lobule has, however, been known to result from an embolism of the first branch of the artery. Very rarely softening is limited to the paracentral region, or to the precuneus. Except the loss of smell that results from disease of the olfactory nerve, the symptoms of softening in the region of the anterior cerebral are not distinctive. Theoretically a monoplegia affecting the leg only should result from softening of the marginal convolution and the paracentral lobule, but I do not know of an observed case.

Middle Cerebral.—Since this artery supplies the motor cortex and the motor path through the ganglia, its obstruction, partial or complete, usually gives rise to paralysis. One or more of its four cortical branches (see Fig. 47, p. 63) may be occluded, or all may be obstructed by a plug at the point of division of the artery at the island of Reil. Occlusion of the branches may cause softening in the area supplied by them, shown in Fig. 48. That of the first produces softening of the third frontal, and, if on the left side, aphasia; of the second or third, softening of the ascending frontal or ascending parietal convolution and hemiplegia, partial when the softening is incomplete; and of the fourth, softening about the posterior limb of the fissure of Sylvius, and if on the left side, sensory aphasia, defective perception of words, with corresponding impairment of speech, are produced. When all the cortical branches are obstructed, the softening extends over all the area supplied, although usually in varying degree in different parts, in consequence of partial anastomoses, and the upper region of the hemisphere may escape (see Fig. 15, p. 23). The usual effect is severe hemiplegia, with impairment of sensibility for a time, and sometimes even affection of the special senses, and ptosis on the opposite side. Partial obstruction of the deep central branches, from the first part of the artery, either by embolism, disease of the wall at their origin, or thrombosis, causes softening in the lenticular nucleus, caudate nucleus, and anterior part of the optic thalamus, very variable in extent; when considerable the internal capsule always suffers, and hemiplegia results. Complete obstruction of the middle cerebral near its origin always causes softening in the central ganglia and hemiplegia, with or without softening of the cortex supplied by it.

Posterior Cerebral.—The rarity of softening from obstruction of the posterior cerebral, due to the freedom of its anastomoses, renders our knowledge of the symptoms produced much less definite than in the case of the middle cerebral. They are for the most part sensory; there may be hemianæsthesia from softening of the tegmentum of the crus, or the internal capsule, or hemianopia from softening of the occipital lobe. Such softening, especially of the cuneus, is the most common cause of cortical hemianopia, but the same symptom is very common in obstruction of this artery when the cortex is intact, from interruption to the optic path. Complete but transient loss of sight has also attended embolism of one posterior cerebral: it is probably inhibitory in origin. The symptoms of limited softenings of the inner and lower part of the temporal lobe are not known.

Basilar Artery.—The symptoms present considerable variations according to the seat of obstruction, whether in the middle, lower, or upper portions, or whether there is merely occlusion of the arteries that come off from it, in consequence of disease of the wall, or of incomplete embolism. In the obstruction of the transverse arteries, the softening produced may be bilateral or unilateral, often small in extent, near the middle line or in the outer part of the pons, according as median or radicular branches are obstructed (see p. 65). Hemiplegia usually results, which may be "alternate," of limbs and cranial nerves, or may resemble that produced by a lesion of the corpus striatum, according to the seat of the disease (see p. 315). In some cases the occlusion of different branches takes place successively, and the patient may have several sudden seizures, with irregular paralysis of limbs and cranial nerves. In occlusion of the whole basilar, the limbs of both sides are usually affected, suddenly or in the course of a few days. The limbs on one side, for instance, may become weak and then improve; a day or two afterwards the other side becomes weak, and then there is again loss of power in the first side. The affection of the facial or fifth nerves may present the same variations, and often does not correspond in degree to that of the limbs. There is usually considerable impairment of articulation and of swallowing. Loss of the conjugate movement of the eyes to one side is occasionally observed, with or without palsy of the facial nerve on the side of the affected external rectus (see p. 188). This, however, less frequently results from softening than from tumour. When the obstruction involves the upper part of the vessel, as is usually the case in embolism, there are ocular symptoms other than the conjugate deviation, ptosis, contracted or dilated pupils, with loss of reflex action to light, sometimes transient. There is occasionally paralysis of the whole or part of one third nerve, or of both, sometimes also of the fourth. Less common is hemianopia, from extension of clot along one posterior cerebral beyond the origin of the posterior communicating artery. Obstruction of the lower extremity of the basilar is not common, except as the result of the extension of a clot from one of the vertebral arteries. The onset of obstruction of the basilar is usually attended with a distinct, but transient apoplectic seizure; it often recurs, and gradually deepens. The patient may lie for some days in a state of incomplete coma, can be roused to open his eyes, but does not speak. Convulsions occasionally attend the onset, but are on the whole not frequent; they may be general or partial; one limb or one side of the face may escape. Rigidity or clonic spasm in the legs or arms is much less common than in hæmorrhage into the pons. Undue frequency and irregularity of the heart's action, and impaired respiration (laboured or sighing, or of Cheyne-Stokes rhythm, or with irregular variations) are very common before death, and may occur from the commencement when the obstruction involves the lower extremity of the basilar, from which the arterioles arise that supply the pneumogastric centre. If there is an apoplectiform onset there may be an initial fall of temperature, much greater than is observed in the occlusion of other vessels, and resembling that met with in cerebral hæmorrhage. In one case the rectal temperature fell to 95°.* Towards the end there is often the rise of temperature so common in hæmorrhage into the pons. In one recorded case the rise reached 109° before death and 109 5° after death. The duration of life is usually from three to seven days; occasionally death occurs within twenty-four hours—in five and a half hours in the case mentioned above, in which the temperature fell to 95°. Rarely life is prolonged for a fortnight. Recovery probably occurs in some cases of syphilitic disease with incomplete obstruction, but not in atheromatous thrombosis, and perhaps not in embolism.

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* Bastian, Clin. Soc., March 13th, 1885.

Vertebral Artery.—The frequent anomalies in the arrangement of the arteries of the medulla render the symptoms of thrombosis of the vertebral artery very variable. It usually supplies the "bulbar" nuclei, hypoglossal, accessory, glosso-pharyngeal, in part directly, and in part by the anterior spinal; and, through the latter, it supplies the anterior pyramids and also the lower part of the medulla. The typical effect of its occlusion is one-sided bulbar paralysis—lips, tongue, palate, and larynx—with paralysis of the limbs on the same side.* The hemiplegia, usually transient, is often accompanied by tingling, and sometimes by anæsthesia. It is transient because the anterior spinal receives blood from the other vertebral artery, and the supply to the lower part of the medulla is renewed. The paralysis of the bulbar nerves, the nuclei of which are supplied in part from the anterior spinal, is usually imperfect, but the impairment of swallowing and of articulation is always great. The median arterioles may come altogether from one anterior spinal (when there are two), and if this is occluded, the bulbar paralysis is bilateral, and may resemble perfectly that from chronic degenerative disease. If, as is often the case, the clot extends up into the commencement of the basilar, the arrest of the blood-supply to the respiratory centre causes rapid death. There may be softening of the posterior part of the cerebellum, the occurrence and extent of which is influenced by the extent of the arterial anastomoses.

Cerebellar Arteries.—These vessels are rarely the seat of isolated obstruction, and although softening may result from occlusion of the arteries from which the cerebellar vessels spring, the softening is always less extensive than the area of distribution of the affected vessel, on account of the free connections,† and any symptoms that are produced are lost in the more serious disturbance that results from the damage to the pons and medulla. Softening is most common in the region supplied by the posterior cerebellar, the obstruction of which is usually secondary to occlusion of the vertebral. Incoordination of movement has been observed in one or two cases of this kind. Occasionally an isolated area of softening is found in the middle of one hemisphere, without any symptoms that could be ascribed to it.

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* The limbs were affected on the same side as the thrombosis in most recorded cases.

† The whole cerebellum can be injected from any one of its arteries, even if the basilar is tied (Duret).

Diagnosis.—The diagnosis of obstructive softening rests on the symptoms of a sudden cerebral lesion, occurring in the conditions that are known to be causes of vascular occlusion. The characters of the attack have less diagnostic significance than the causal indications. The chief difficulty is the distinction of atheromatous obstruction from hæmorrhage. Under forty, hæmorrhage would only be suspected if the apoplectic attack were one of considerable severity, and the coma prolonged; a cause of aneurism, heart disease, syphilis, or injury can then usually be traced. In most cases during the first half of life the apoplectiform symptoms are moderate, slight, or absent, and one of the two common causes of vascular obstruction can be traced—heart disease or syphilis. Usually one is present and the other absent, and the diagnosis can then be made with confidence. If both are present, *e. g.* if the patient is suffering from heart disease and has had syphilis, the diagnosis is much more difficult, and rests, first, on the evidence of activity of one or the other of these causes, and secondly, on the presence of any other symptoms that may be due to syphilitic disease of the brain. If there is considerable valvular mischief, if there has been recent endocarditis, and especially if evidence of embolism elsewhere can be discovered (as an enlarged and tender spleen, or sudden hæmaturia, or embolism of the central artery of the retina), the probability of cerebral embolism is very great. On the other hand, persistent headache for some days or weeks before the onset, transient attacks of tingling, numbness, or weakness in the limbs afterwards paralysed, or a dull somnolent state before the attack, suggest syphilitic disease of the arteries. Evidence of recent activity of the syphilis adds weight to the other symptoms, but is not often forthcoming, since the period at which arterial disease occurs is later than that at which the obtrusive manifestations of constitutional syphilis are common. Apparent quiescence of the constitutional disease is thus no contra-indication, nor is past antisyphilitic treatment, however thorough. This is a frequent source of error. Hence, before the degenerative period of life, in the absence of other causes, syphilitic disease is probable, unless the ordinary mode of infection can be absolutely excluded. As an instance of the difficulty the diagnosis sometimes presents, I may mention the case of a man who had an attack of hemiplegia at forty-five. He had a loud aortic regurgitant murmur, and it seemed probable that the hemiplegia was due to embolism, with which the onset was consistent. But he had had severe headache for some weeks before the attack. Shortly afterwards a node appeared on his skull, and it was ascertained that in youth he had had syphilis. This fact gave significance to the headache, and made it far more probable that the vascular obstruction was due to syphilitic disease than to embolism.

A year or more after the onset, the absence of heart disease does not exclude embolism, or even render it less improbable, if the hemiplegia occurred during or soon after an illness known to cause endocarditis, especially rheumatism or chorea; but without such disease, or soon after the onset, embolism is unlikely if no source can be found. A girl had an attack of chorea, attended by endocarditis and mitral regurgitation. While still ill with the chorea she had an attack of hemiplegia, no doubt due to embolism. Some years later, the hemiplegia persisting, her heart presented no abnormal sign in sound or impulse.*

During the degenerative period of life, after forty-five, the diagnosis of softening presents much greater difficulties. The help afforded by causal indications is more limited. The conditions associated with hæmorrhage and softening are to a large extent the same. Atheroma, the chief cause of softening, is met with in four fifths of the cases of hæmorrhage. The state of the heart is of more significance than that of the vessels. A strongly acting, hypertrophied heart suggests hæmorrhage; a feeble, irregular heart, softening: the former tends to burst a weak vessel, the latter to permit clotting in one that is diseased. The causes which predispose to thrombosis by influencing the blood and circulation are also of diagnostic importance. Thus gout, cancer, phthisis, prostration, or mental depression are all in favour of this lesion. Other diagnostic indications are drawn from the characters of the onset, but these have been already described in the chapter on hæmorrhage. It must be remembered that the diagnosis is always a matter of probability, sometimes high and sometimes low, but it is seldom that the indications are so equally balanced that a probable diagnosis cannot be made.

Still more complicated, however, are the cases in which more than one cause of a vascular lesion of the brain can be traced, but the additional complexity relates to the cause of softening rather than to the distinction from hæmorrhage. The latter is indeed facilitated, since the presence of more than one condition capable of causing softening increases the probability, *cæteris paribus*, that the lesion is not hæmorrhage. But the question which of several causes of vascular obstruction has been effective in a given case can only be answered by a careful consideration of the apparent activity of the several causal influences, and by the symptoms at the onset as already described. Atheroma and syphilitic disease may both be probable, and they have more symptoms in common than either has with embolism, since in each there may be interference with the circulation, and often therefore slight symptoms before the onset. Moreover, the co-existence of heart disease with the causes of thrombosis introduces another element of uncertainty, since the same condition of the circulation favours the formation of clot in the vessels of the brain and on the valves of the heart. This is true both of syphilitic disease of the arteries and of atheroma, and illustrates the law that in all cases of merely probable diagnosis, the rules that lead to a correct opinion in most cases, and must therefore be followed, now and then fail us. A woman, aged sixty, was brought unconscious to the hospital with symptoms that pointed to obstruction in the basilar artery. Her vessels were highly degenerated, and she had a loud murmur of mitral constriction. Of the two lesions that seemed possible, atheromatous thrombosis and embolism, the rarity of the latter in the basilar artery made the former rather more likely. She died, and thrombosis of the basilar was found as anticipated, but it was due, not to atheroma, but to intense and characteristic syphilitic disease of the vessel, which there was nothing during life to suggest. Another example of a like difficulty has just been mentioned.

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* Yet several years later she presented signs of mitral constriction, a most significant fact, although not strictly relevant to the present subject.

Prognosis.—The prospect of recovery from the initial symptoms depends on their severity and duration, on the previous occurrence of similar attacks, on the artery affected, and on the nature of the cause. Intensity of apoplexy is a graver indication than its duration. In this respect softening differs from hæmorrhage. It is not uncommon for a patient to recover after what may be called "comatose stupor" has lasted for five or six days; but deep coma, with impairment of respiration, is as rarely recovered from in softening as in hæmorrhage. The stage of inflammatory reaction is attended with considerable danger in severe cases, and the occurrence of rapid sloughing or of convulsions is usually of fatal augury.

It is rare for a patient to die in a first attack of cerebral softening unless a very large vessel is occluded. But if the brain has been damaged by a preceding attack, recovery occurs less readily, and the immediate danger to life increases with the number of preceding attacks, especially in the old, or when there is extensive syphilitic disease, or wide-spread atheroma.

Thrombosis of the internal carotid or of the basilar entails imminent danger to life; indeed, the latter, from whatever cause it arises, is rarely survived. Next in gravity is obstruction of the vertebral, and next that of the middle cerebral. Obstruction of the trunk of both middle cerebrals is almost always fatal. Subject to these indications, the danger to life is far less in embolism and in syphilitic disease than in senile softening. In most fatal cases of cerebral embolism death has been due rather to the state of the heart or to some general disease, as rheumatism. Syphilitic disease rarely causes death unless the basilar artery or many vessels are affected, or the nature of the malady is not recognised and met by appropriate treatment.

The prognosis as to recovery from the persisting symptoms depends on the seat of the disease, and the reasons for regarding the symptoms as "direct" or "indirect" in nature. These indications are the same as in cerebral hæmorrhage, and are described in the account of this (p. 396), and also in the description of the individual symptoms. The prospect of recovery depends also to some extent on age. Recovery of slightly damaged parts, and compensation by other parts, take place more readily in the young than in the old. But the prognosis as regards recovery is but little influenced by the nature of the disease. It is no better in syphilitic disease than in embolism. We may, by treatment, remove the disease of the wall of the occluded vessel, we may prevent an increase in the symptoms, but we cannot remove the clot that has finally closed it, and has extended on into the distal branches of the vessel. We cannot, therefore, restore the circulation through the vessel. Neither can we influence, by antisyphilitic treatment, the process of softening, which, in syphilitic disease, just as in embolism, is a process of simple necrosis. Therefore the syphilitic origin of the disease does not influence the prognosis of developed palsy. Most cases improve, and many recover, but they improve and recover in the same way as in every other form of acute cerebral lesion—because the symptoms are of indirect and not of direct origin, and sometimes because a collateral circulation is possible. If the softening involves the motor path or centres, and no compensation is possible, enduring paralysis is the result. It is necessary to insist strongly upon this fact. Because a palsy is due to syphilis, it is often assumed, as a matter of course, that it will be removed by antisyphilitic treatment. The assumption is correct as regards many pressure-effects of syphilitic growths, and many syphilitic inflammations, but it is not true of necrotic softening from vascular disease. I have seen many patients who had been assured that, because their hemiplegia was of syphilitic origin, they would certainly be cured; and when, after a year or two, the paralysis remained, they were naturally indignant at the erroneous opinion they had received.

It must be remembered that convulsions at an interval *after* the onset are prone to recur, and the tendency to the return is greater the longer the interval. They may continue even when the paralysis has passed away, and may constitute a disease resembling epilepsy in its course. Such recurring convulsions are far more frequent after softening than after hæmorrhage, partly because the cortex is more often involved.

The probability of a recurrence of paralysis varies according to the nature of the disease. In embolism it is not great. Although a second attack is possible, and instances are occasionally met with, it is a rare accident, and in any given case a recurrence is unlikely. In syphilitic disease the probability of a recurrence depends on proper and repeated treatment (see "Syphilitic Tumour"), and also on the previous duration of the disease. The arterial walls may be so profoundly altered, that treatment can only induce a cicatricial state so abnormal that coagulation is favoured. But the nature of the case is now usually recognised and treated early, and hence a recurrence of softening is extremely rare. In atheromatous softening, on the other hand, the tendency to recurrence is very great. Many arteries are always affected; the disease of their walls is, for the most part, beyond control, and sooner or later other vessels become obstructed.

Recurrence is more likely to be speedy—*e. g.* within two or three years—if the first attack occurred independently of influences favouring thrombosis, than if it was "excited." If, for instance, the attack came on after depressing grief, during a condition of general illness, or in consequence of prostrating fatigue, and these influences are avoided, a recurrence is more likely to be long deferred. This is also true of states of the blood which are under control. On the other hand, a speedy recurrence is more probable if there are conditions favouring thrombosis that cannot be removed, especially cardiac weakness and dilatation. Exceptions to these rules naturally occur, because we cannot tell the extent and degree of the vascular disease, and this may sometimes invalidate conclusions that are reached by a process which usually leads to a correct opinion.

TREATMENT.—The treatment of the initial apoplexy is in part the same as in cerebral hæmorrhage; in part it is different. The same precautions are necessary as to the avoidance of movement, and of any hindrance to the return of blood from the head. Sinapisms to the back of the neck seem to hasten the recovery of consciousness, and, by causing reflex contraction of the arteries, they probably quicken the cerebral circulation and lessen the tendency to stasis. But whereas in hæmorrhage the object of treatment is to lower the blood-pressure and promote coagulation, in softening the aim is to lessen the tendency to clotting, and to increase the flow of blood at least up to normal conditions. The action of the heart should be kept uniform and strengthened if necessary by small doses of digitalis or strophanthus. By doing this we may lessen the tendency to the spread of the thrombus (whether this is primary or secondary). The patient should lie with the head and shoulders slightly raised. The bowels should be opened, but more gently than in hæmorrhage, because violent purgation, weakening the heart, may increase the tendency to an extension of the clot. If the patient is gouty and the heart regular, a somewhat stronger aperient may be given, which should contain some mercury. Under the same circumstances a diuretic is useful—half-drachm doses of nitrous ether by the mouth or by the rectum. Venesection is not to be thought of in any case that may possibly be of this nature. Whether stimulants are given or not must depend on the state of the pulse and heart. If indicated, ether, ammonia, or alcohol may be given, the latter more freely than in hæmorrhage. If there is doubt whether alcohol should be given or not in hæmorrhage, it is better to withhold it; in softening it is better to give it.

After the patient has regained consciousness, mental and physical tranquillity is of the first importance. All excitement and movement should be carefully avoided, since whatever disturbs the heart's action is liable to be followed by reactionary failure that will favour extension of the thrombus. Food should be light, easily digested, and nutritious. Alcohol is better avoided after the first two or three days (unless the state of the pulse demands it), on account of the risk of increasing the intensity of the inflammatory reaction. During this stage the management of the case requires great care, and should be conducted on the same principles as a local inflammation elsewhere. The diuretic given at the onset may be continued. The bowels must be kept carefully open. The patient's head may be raised a little higher, but the shoulders should always be raised as well as the head. If there is headache and fever, ice may be applied to the head, and moderate doses (10 or 12 gr.) of antipyrin may be given. Leeching has been advocated, but it is safer to abstain from any form of depletion, unless the patient is manifestly plethoric. If convulsions occur during this stage, they are often arrested by the measures recommended for the headache, and moderate doses of bromide may be given. The same treatment is suitable for delirium. Extreme cleanliness is essential; it is at this stage that bedsores are most apt to form. The limbs and back should be daily examined, and on the least indication of trophic disturbance the patient should be placed on a water-bed, or the pressure be kept from any reddened spot by pillows or cotton wool. The latter, charged with some disinfecting agent, is especially useful when there is any escape of urine. After the inflammatory stage is over, tonics are usually needed, especially iron and nux vomica or strychnine; but the measures likely to lessen the tendency to further thrombosis must be continued, and the state of the pulse, heart, urine, &c., should be frequently observed.

The variations in this treatment which the special form of obstruction renders necessary are not great. In embolism the general principles of treatment are also in the main the same as in thrombosis, because the latter process is always associated with embolism, clot forming not only on the distal side but also on the proximal side of the obstruction up to the origin of the first large branch, and it is important to avoid whatever may facilitate the extension downwards of this secondary clot. Moreover, it is important in embolism as in thrombosis to strengthen and steady the action of the heart, in order to lessen the risk of further embolism. This is more likely to occur if the heart acts irregularly, because vegetations tend to increase during the feeble action, and to be detached when it becomes excited. But, in syphilitic disease, although treatment is powerless to undo mischief that has already occurred, it may prevent its increase or fresh lesions. Many arteries are usually affected; the same artery may be diseased at separate places, and it is of the utmost importance to remove this disease. Iodide of potassium should therefore be given in doses of seven or ten grains every six or eight hours, or fifteen-grain doses may be injected into the rectum. It may be well also to rub in some mercury, especially if the symptoms indicate extensive disease. The importance of prompt treatment cannot be too strongly urged, because the closure of another artery may be prevented, or very early symptoms, due to commencing thrombosis, may certainly be arrested. But it is not well to give iodide in larger doses,* or to employ it in other forms of thrombosis, because it has a distinct tendency to promote coagulation, as its value in aneurisms testifies.

When treatment fails to avert further thrombosis, it is because the arterial walls are so profoundly changed that it is not possible for them to regain a normal state. Nor is it well to continue the treatment for more than six or eight weeks. In that time it will have achieved its objects: persistent symptoms are due to the disease of the nerve-elements, and may or may not slowly pass away. But the treatment should be renewed for three weeks every six months, for at least three or four years, to arrest the development of commencing disease, probably by preventing insusceptible germs from becoming susceptible and injurious organisms.†

The physical rest that is needed must vary with the severity of the disease, and may be two weeks in slight cases, and four or six weeks in those that are more severe. When the stage of inflammatory disturbance is over, and the process of repair of the less damaged structures has commenced, nervine tonics may be given, such as quinine, strychnia, hypophosphite of soda, and other tonics that may be indicated, such as iron. For the reasons to be mentioned presently, it is impossible to obtain satisfactory evidence that nervine tonics do good, but it is probable that they have some influence in the right direction.

For the treatment of the residual symptoms that depend on the destruction of tissue, little, unfortunately, can be done. It is convenient here to describe the general treatment of hemiplegia, since the measures suitable to that which is produced by softening are the same as in other lesions. Many of these effects lessen after a time, with and without treatment, in consequence of the compensation effected by other parts of the brain. In hemiplegia, for instance, improvement always occurs in the leg, even when the motor path to the affected side is completely interrupted, in consequence of the other hemisphere gaining that power over the limb for which anatomical arrangements always exist. The indirect symptoms pass away spontaneously.

In the early stage of hemiplegia, gentle rubbing of the limbs is all the local treatment admissible. It has a tendency to lessen the rigidity, and may be continued and made more vigorous when the early rigidity has passed away and the late rigidity has set in. Upward rubbing of the muscles has most influence in lessening the spasm. The influence of electricity on the paralysis is very small, as may, indeed, be anticipated from the nature of the palsy. Late rigidity in the flexors is lessened, for the moment, by faradisation of the extensors, and repeated applications sometimes seem slightly to diminish the contraction. If there is some power of voluntary movement, the stimulation of the muscles by any form of electricity is followed by a slight temporary improvement in power, but the improvement is not permanent, and it is doubtful whether any lasting benefit results from a course of such treatment. In any case it is not desirable to apply electricity to the limbs during the first six weeks after the onset. The stimulation of the sensory nerves of the limbs has an influence on the brain, as the arrest of a commencing epileptic fit by such means clearly shows, and the early application of electricity has been followed immediately by a fresh attack of paralysis. Any influence on the cerebral disease, of the voltaic current applied to the heart, is not within the range of therapeutic possibility. The tendency to improvement by cerebral compensation, and by the spontaneous disappearance of indirect symptoms, is very marked, and makes it difficult to estimate the actual influence of treatment that is employed, especially of methods that extend over a long period, and thus have time on their side; moreover it renders these cases a tempting field for the assumptions of the *quasi*-therapeutist. It is most important, when there is evidence of destruction of the motor path, that the friends of the patient, if not the patient himself, should be made aware of the hopelessness of a search after a "cure," on the one hand, and, on the other, of the slow improvement that time will bring.

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* Unless unwise persistence has habituated the patient to it, so that it no longer checks the syphilitic lesions when given in ordinary doses, to which the organisms of the disease seem to get accustomed. (See next note.)

† See the writer's lectures on 'Syphilis and the Nervous System,' London, 1893.

In addition to the measures just mentioned which tend to lessen late contractures, mechanical means are sometimes necessary for cases in which it becomes extreme in degree. The hand may be placed on a splint for a few hours each day; the spasm may be readily overcome, so as to apply the splint, by gentle continued extension, aided in severe cases by the immersion of the limb in warm water. A small cylinder may be placed in the hand within the flexed fingers, or an india-rubber ball to which a tube is attached may be introduced, and inflated after it is in the hand. When the paralysis of the leg is considerable, there is often shortening of the calf muscles, due to the weight of the foot, which brings it into the position of extension as the patient lies. This is a serious inconvenience, because it may prevent the patient standing and walking when he acquires sufficient muscular power. It can be more readily prevented than cured. The foot should be carefully kept at right angles to the leg during the early stage, by a footboard, or by a sand-bag, about nine inches in diameter, beneath the sole. At a later period it may be necessary to keep the foot up by attaching a cord, with a spring inserted in it, from the front of the boot to the knee, or by the use of a suitable splint. The active character of the contracture renders tenotomy undesirable.

The post-hemiplegic athetoid spasm is very difficult to influence by treatment. Electricity fails, as a rule, to lessen it. I have, indeed, met with one case in which the spasm presented a very marked and permanent diminution during a course of treatment with the voltaic current, but no such effect was produced in a number of similar cases in which I employed the same treatment, and I am therefore disposed to regard the improvement as a coincidence rather than as a consequence. The inco-ordination is somewhat lessened by the persevering practice of hand gymnastics—rhythmical movements of the fingers and thumb. A set of "dumb piano" keys is convenient for this purpose. These are made for exercising the fingers of pianists, and are so constructed that the resistance of the keys can be varied by a spring. A type-writer is useful for slighter cases, and trains the brain as well as the hand. Systematic rubbing has also a trifling influence on this as on other forms of inco-ordinate spasm.

Softening of the brain is the most common cause of aphasia, and the question often arises whether any special treatment should be adopted for the loss of speech. So far as concerns general therapeutic measures, the treatment for the condition of the brain already mentioned is all that can be done; the only special treatment for the loss of speech is educational. The question only arises in a severe case of speech defect; slight defects pass away without more training than ordinary life supplies. But if there is destruction of the speech region, motor and sensory, on the left side of the brain, speech is recovered by a process of training of the right hemisphere so as to bring the right-sided speech-processes into a relation to the will similar to that of the processes in the left hemisphere. The readiness with which this can be effected varies in different individuals. In some it occurs speedily, in others it never occurs, and all treatment fails to evoke any power of voluntary speech. In the former, systematic training hastens the compensation. Possibly it is facilitated by the use of the left hand for working and other objects. The precise method to be adopted must depend on the character of the defect, consisting in the exercise of the speech-process that is deficient. If there is word-deafness, simple directions must be uttered to the patient, and gradually varied and made more complex; if the patient has a difficulty in reviving word-images, he must be exercised in naming objects; if there is motor aphasia without word-deafness, he should try to repeat words after another person. So with inability to read and to write. In motor aphasia, attempts to write are useless until the patient has made some progress with articulate expression. In every case the exercises must begin with the simplest word-processes of each kind, and these should be mastered before more difficult processes are attempted. The practice should not be continued long enough to fatigue the brain, but should be repeated several times a day. Patience and perseverance are needed, but will meet with their reward.

THROMBOSIS IN THE CEREBRAL SINUSES AND VEINS.

Coagulation of blood may occur in the cerebral sinuses and veins, and may cause mechanical congestion, œdema, capillary hæmorrhages, and sometimes softening, in the parts from which the blood should be conveyed by the occluded vessel. The occurrence is usually of very serious significance, on account not only of its effects, but also of the gravity of the conditions with which it is associated. In order to understand the pathology of the condition, it is necessary to bear in mind the chief facts regarding the venous circulation of the brain, already described at p. 60.

CAUSES.—Sinus-thrombosis may occur (1) from the state of the blood and circulation generally; (2) in consequence of disease adjacent to the sinuses. Having regard to the local process itself, the former may be called *primary*, the latter *secondary* thrombosis. The secondary thrombosis is more than twice as common as the primary.

1. *Primary thrombosis* usually occurs in association with general malnutrition and prostration, and hence is often termed "marantic thrombosis."* It occurs at all ages, but most frequently in children, and next in frequency in the very old. In children, it is met with up to fourteen years of age, but the chief liability is during the first six months of life, and the chief cause is severe and exhausting diarrhœa. It may, however, follow almost any prostrating malady, lung disease, long-continued suppuration, or acute specific diseases. In adults, it occurs occasionally during the last stage of phthisis, sometimes from acute diseases, in the puerperal state, as a result of gout, or in the course of cancer.

These causes no doubt act partly by weakening the heart and so retarding the circulation, and partly by rendering the blood more prone to coagulate. Diarrhœa may act also by reducing the volume of the blood. The rigid walls of a sinus cannot contract in adaptation to the lessened volume of the blood, and hence the circulation in it must be unduly retarded. The trabeculæ that cross its cavity doubtless also facilitate the formation of a clot, and so also does the irregular shape of the cavity. The seat of primary thrombosis is almost invariably the superior longitudinal sinus, very rarely the lateral, but occasionally, in old persons, the cavernous sinus. It is probable that the current of blood in the superior longitudinal sinus is normally more feeble than in any other vessel of the body. The veins that it receives, open into it in a forward direction, so that the motion of the entering blood is opposed to that of the blood in the sinus. Moreover, these veins ascend to the sinus, and nowhere else in the body do ascending veins receive the blood of ascending arteries. Destitute, therefore, of the influence of the hydrostatic pressure so potent elsewhere, the blood within them is only influenced by the force of the heart, exerted through the capillaries. It is, therefore, not surprising that thrombosis should readily occur in this vessel, when the circulation is still further weakened by disease. This result is favoured by the irregularity, not only of the shape, but of the inner surface of the sinus, which is such that a layer of a clot is sometimes found upon it, although a channel remains for the passage of the blood.

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* The term marasmus is currently used in a more restricted sense than is implied in this use of the adjective, although the latter is justified by the original meaning of the word.

Does primary thrombosis ever occur in the veins and not in a sinus? There is some pathological evidence that it does. Young children are sometimes seized with hemiplegia, commencing with unilateral convulsions that are prone to recur. Sometimes the attack comes on without exciting cause, sometimes under the same conditions as sinus-thrombosis. The lesion which causes the symptoms is often cortical, and its ultimate aspect is consistent with the theory that it was due to thrombosis in one of the veins of the convexity. Very similar symptoms in the course of tubercular meningitis have been proved to be due to thrombosis in a vein, and the same process has been met with in the subjects of phthisis without inflammation or visible tubercle.

2. *Secondary thrombosis* is the result of disease adjacent to a sinus. The most common cause is caries of the bone, and especially disease of the internal ear. Other causes are injury to the bone (especially when attended with inflammation of the diploë), meningitis, especially tubercular, compression of a sinus by a tumour or foreign body, and suppurative diseases outside the skull, erysipelas, carbuncle of face or neck, malignant ulceration, and even, it is said, suppurating eczema of the scalp (Tonnelé). This form occurs with nearly the same frequency throughout life, except that it is rare in the very young and in the very old.

The mechanism by which local disease acts is probably threefold. First, the inflammation may extend to the wall of the sinus, and the inflamed wall may cause coagulation of the blood within it. Secondly, a clot may extend along a vein that passes from the seat of the disease to the sinus. Almost every sinus receives veins from the exterior of the skull (see p. 67), many of them also from the diploë, while the superior petrosal and lateral sinus receive blood from the internal ear. The third mechanism is the simple compression of a sinus, but this is very rare. The first and second are common, and it is often difficult to say which has been effective in a given case. There is sometimes distinct evidence of inflammation of the wall of the sinus, and meningitis frequently co-exists. But in other cases the wall of the sinus is healthy, and the clot has apparently extended into it by prolongation. Even when there is inflammation of the wall, this may be, in some cases, secondary to the formation of the clot. Hence it is not desirable to call the whole of this group "phlebitic thrombosis." The sinus affected is always that nearest to the local disease, and, as ear disease is the most common cause, the most common seat of thrombosis is the superior petrosal or lateral sinus. The superior longitudinal sinus is very rarely the seat of a secondary thrombus. In tubercular meningitis and phthisis the clot may be limited to a vein.

Pathological Anatomy.—The affected sinus is usually filled with a clot adherent to the walls, the more firmly the older it is. A recent clot is dark red and soft, but after a few days becomes paler, granular, and friable. It is usually laminated. Sometimes the clot does not entirely fill the sinus. When due to adjacent caries, it is usually softened and puriform in aspect, and often contains pathogenic organisms. The clot may be limited to one part of the sinus, or may extend through its whole length, and even beyond it, into the tributary veins, or into another sinus, and even (in the case of the lateral sinus) into the commencement of the internal jugular vein. The wall of the sinus is rarely inflamed in marantic thrombosis, but often when the condition is due to the extension of adjacent inflammation of septic character. Meningitis is common under the same circumstances. The veins from which the sinus receives its blood are always greatly distended, and the coagulation may have spread into one or more. The mechanical obstruction to the return of the blood causes intense congestion and œdema in the part from which the tributary vessels come. Capillaries often give way, especially in the grey substance, so that this is crammed with minute points of hæmorrhage. This is most marked when and where the clot has extended into the tributary veins. If the clot is limited to a sinus, some blood can pass from the veins by other channels; but if a vein is plugged, the circulation in the part is arrested. The result of the œdema and hæmorrhage is to cause softening, often limited to minute foci, but sometimes diffuse, so that a mulberry-coloured pulp results. Very rarely there is meningeal hæmorrhage. If the patient recovers, the destruction of tissue is rarely so great as in arterial obstruction, and the affected part of the cortex ultimately presents atrophy and induration of the convolutions. When the clot softens, foci of inflammation may often be found in the lungs, sometimes purulent, constituting secondary septic abscesses. They are present in about half of the cases of secondary (septic) thrombosis.

Symptoms.—The symptoms vary much, and are often masked by those of the condition that gives rise to the thrombosis or results from it,—the cerebral anæmia in primary thrombosis, the adjacent disease or the meningitis it excites in the secondary form. The symptoms directly due to the thrombosis are of two kinds,—those produced by the interference with the functions of that part of the brain in which the circulation is obstructed, and those due to the disturbed circulation outside the skull in the parts from which veins pass through the bone to the internal sinus. The former symptoms have little in themselves that is characteristic; and the latter, although characteristic, are comparatively rare.

Symptoms of Thrombosis of Special Sinuses: Superior Longitudinal.—External œdema and distension of veins may be present on the sides of the head and forehead. There may be epistaxis from the veins of the nose, which communicate with the anterior extremity of the sinus. In young children, the fontanelle may become prominent, in striking contrast to its previous depression if there had been collapse from diarrhœa. Cerebral symptoms are chiefly general—apathy, somnolence, and coma; vomiting and convulsions, usually general, but sometimes local; rigidity of the neck, and sometimes of the muscles of the back. In adults, convulsions are less common than delirium, quiet or active. There is usually headache. In both children and adults there may be strabismus, tremor of tongue or limbs, rigidity of all the limbs, or of the arms only or the legs. Unilateral symptoms are usually due to the extension of the thrombus into veins over one hemisphere, and then there may be unilateral convulsions, often beginning locally, and loss of power on one side, sometimes limited in extent. Thus, in a man in the last stage of phthisis, the superior longitudinal sinus contained an old, granular, partly discoloured clot, which did not quite fill it, and a more recent clot had extended into the veins over the right frontal lobe, which was intensely congested with capillary hæmorrhages. The symptoms were mental dulness for a day or two, and then repeated convulsions, each beginning in the left arm, and involving the whole of the left side, then ceasing on the left side and involving the right. They recurred every ten minutes until death two hours later. Such symptoms, if initial, suggest that the thrombus began in the vein.

Cavernous Sinus.—There is usually some œdema about the eyelids and temples, and enlargement of the veins about the orbit, in consequence of the communication of the ophthalmic and facial veins. There may be distension of the retinal veins, usually transient, and slight œdema of the optic papilla. The eyeball may be prominent from the distension of the orbital vessels. Headache is common, but unilateral limb symptoms are rare. Sometimes there is paralysis of the nerves to the orbit which run in the wall of the sinus, including the first division of the fifth. Bilateral symptoms have resulted from thrombosis of both sinuses. Neuro-paralytic ophthalmia has been observed (Lebert).

Petrosal Sinuses.—It is doubtful whether distinctive symptoms attend thrombosis of either of the petrosal sinuses.

Lateral Sinus.—There may be distension of veins and œdema over the mastoid process, and the cause of the thrombosis, ear disease, is usually obtrusive. The cerebral symptoms are not distinctive, since they resemble those of local meningitis.

Symptoms due to the Nature of the Thrombosis.—In *primary* marantic thrombosis the *symptoms vary* in their accentuation. In adults, the symptoms of a cerebral lesion of some kind are usually distinct; the significance of the somnolence and coma, of the delirium and headache, or of the motor symptoms, is unmistakable. But in young children, the simple exhaustion from the diarrhœa is often attended by somnolence and inertia hardly less severe than that which attends the occlusion of the sinus. Epistaxis, œdema of the scalp, or retraction of the head, under such circumstances, should always attract attention. The temperature is normal, or raised only one or two degrees.

Secondary.—The phlebitic form is usually due to ear disease, and the sinuses most often occluded are those near the ear: the blood is able to escape by other channels; the damage to the brain is therefore slighter, and the direct symptoms are less obtrusive than in thrombosis of the superior longitudinal sinus. Indeed, the cerebral symptoms that occur are due rather to the meningitis which usually coincides than to the closure of the sinus. The clot that forms is usually septic, and breaks down quickly into purulent infective material; hence the symptoms of septicæmia are often more pronounced than are those of intra-cranial disease. In some cases the local symptoms have been so slight that the occurrence of a morbid process was not suspected until the signs of secondary pulmonary abscesses were found. More frequently the septicæmic symptoms are of considerable severity,—rigors, remitting or intermitting pyrexia, and a typhoid aspect. Gradually, cerebral symptoms become more prominent; there is headache, sometimes local. The patient becomes dull, somnolent, and comatose; or, on the other hand, there may be delirium, quiet or violent. Motor symptoms may consist in varied paralyses, disordered sensation, convulsions, or rigidity, due chiefly to the meningitis. Their significance depends on their association with septic symptoms, or with the subcutaneous œdema already described. When the secondary thrombosis affects other sinuses, the special local symptoms already described may be more pronounced.

The onset of the symptoms is sometimes sudden, but more often insidious. Their duration varies from a few days to a fortnight. Coma usually precedes death. Occasionally severe convulsions may exhaust the patient in a few hours. Recovery is extremely rare when the external symptoms leave no doubt of the accuracy of the diagnosis,* but it sometimes occurs in marantic cases. It is probable that the clot may be absorbed and circulation re-established; but if the thrombus has extended into a vein, permanent damage to the cortex may result.

DIAGNOSIS.—In marantic thrombosis in the adult the occurrence of cerebral symptoms may lead to a suspicion of the condition if the patient has no heart disease and has not reached the degenerative period of life, but the diagnosis is only certain when there is external œdema or distension of veins. In an old person, without these symptoms, the cerebral disturbance is more likely to be due to thrombosis in an artery than in a vein. In young children, somnolence, coma, and even general convulsions may be due merely to cerebral anæmia (in the state termed "hydrocephaloid"), but if these symptoms continue after the diarrhœa has ceased and the collapse is slighter, thrombosis is probable; the occurrence of local brain symptoms increases very much the degree of probability, and the addition of the external symptoms renders the diagnosis certain. In phlebitic thrombosis the diagnostic difficulties are much greater. The cerebral symptoms are due to and suggest meningitis rather than thrombosis, and the indications of the latter are chiefly those of septicæmia (due to the softening of the clot) and especially the external œdema.

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* An instance of recovery with some permanent damage to the left hemisphere is recorded by Voorman ('Deut. med. Wochenschrift,' 1882, No. 36) in a child four months old, in whom the symptoms were tremor of tongue and right limbs, rigidity of neck, opisthotonos, with distension of the left temporal vein, and œdema of the scalp.

Prognosis.—This is extremely grave in all cases. Phlebitic thrombosis with septicæmia is probably always fatal. Marantic thrombosis is occasionally survived in children, but scarcely ever in adults, in whom it usually supervenes on a disease that is itself incompatible with the long continuance of life. If a patient recovers, the general symptoms pass away, but local symptoms may persist, and there may be some impairment of intellect. Possibly some cases of so-called atrophy of the brain, that supervene on an acute illness in childhood, have this origin.

Treatment.—In cases of primary thrombosis the most important elements in treatment are to arrest the cause of the prostration, and to maintain and increase the strength of the circulation by stimulants and tonics such as bark. The posture should be that which will aid the flow of blood in the special sinus affected—if it is the superior longitudinal, for instance, flexion of the neck should be carefully avoided. No depleting measures are permissible.

In secondary thrombosis, treatment usually fails to exert any influence on the disease. Free exit for all discharges from any wound must be secured. For the septicæmic form, quinine and salicylate of soda have been recommended. I think that full doses of tincture of the perchloride of iron more frequently produce a distinct effect upon this condition than any other drug, but it may be doubted whether recovery ever results from the use of internal remedies only. Surgical measures have, moreover, been successfully employed, even in cases due to ear disease. Ballance has opened the sinus after (double) ligature of the jugular vein, and has cleared out the thrombus with a satisfactory result, and without extension to the opposite sinus.* Sulzer has done the same, including the removal of necrosed dura mater over the sinus, outside which was a collection of pus.[*]

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* Ballance states that the left sinus opens into the straight sinus, and the right into the longitudinal sinus (Ballance, 'Lancet,' 1890, vol. i).

ACUTE CEREBRAL PALSY OF CHILDHOOD (INFANTILE HEMIPLEGIA).

Hemiplegia of sudden onset is not uncommon in children, especially in young children.[†] There is considerable difference of opinion as to the exact pathological condition on which it usually depends, and it is probable that the cause is not always the same. Hence it is convenient to give a brief account of the condition as a clinical variety of disease. It is probably not a distinct pathological variety. Many of the cases are examples of one or other of the forms of disease already described. In almost all cases there is paralysis—hemiplegia. Sometimes, perhaps, a lesion occurs and causes no local symptom, but such cases can scarcely be recognised. Very rarely there is general loss of power, but it is probable that in such cases both hemispheres are diseased: there is double hemiplegia.

It is to be understood that only the cases in which the onset is acute or sudden are now considered. Hemiplegia of chronic onset is generally due to a cerebral tumour. Another class of cases which must be excluded are those in which the paralysis dates from birth, and is due to injury received during the process of birth. These cases have been considered in the section on meningeal hæmorrhage.

The following account of the acquired palsy is based on an extensive and consecutive series of cases that have come under my observation.

Acute cerebral palsy with a distinct onset during infancy or childhood is rather more common in females than in males, the proportion being about five to four. In three fifths of the cases the onset was during the first two years of life, three quarters during the first three years, and seven eighths during the first five years. It seems to be equally common in the first and second year of life. Males preponderate during the first year, and females after the first year.

In the majority of the cases the disease is not distinctly secondary to any morbid influence; it was apparently primary in five of the eight cases. In some of these the onset was during hot weather, and in a few there had been distinct exposure to the sun, but the relation to season was not investigated in a sufficiently large number to justify any conclusion. We must remember that, by mere coincidence, nearly a third of the cases may be expected to occur during the time of the year when hot weather is occasionally met with. The onset sometimes occurs within a few days of a severe fall, especially during the first two years of life. It may also be secondary to pneumonia and to severe diarrhœa, chiefly in early infancy.

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[*] 'Wien. klin. Wochensch.,' 1890.

[†] The condition has been carefully studied by Strümpell, and valuable analyses of cases have been published since the first edition of this work appeared by Osler ('The Cerebral Palsies of Children,' 1889) and Sachs (see reference on p. 413, and in "Die Hirnlahmungen der Kinder," 'Volkmann's Samml. Kl. Vorträge,' 1892). Most of the facts ascertained by them are, however, embraced in the briefer account which follows.

Of other diseases to which the condition was distinctly secondary, two acute specific diseases take the first place—scarlet fever and measles. In almost all, the onset was during the decline of the acute specific disease, or within a fortnight of its termination. In some of the cases after scarlet fever, dropsy was present at the time of the onset; in others there was no dropsy. The cases after scarlet fever occur chiefly between two and five years of age; those after measles between one and four. The malady may also come on during severe and prolonged whooping-cough or bronchitis, and in later childhood during the course of "gastric fever." It very rarely comes on during rheumatic fever, but I have known it to follow mumps.

The onset was attended by severe convulsions in more than half the cases; in some the convulsions recurred at short intervals during several hours, and at the end of that time the child was found paralysed. In other cases, several distinct attacks of convulsion were separated by hours or days, and the hemiplegia was only found after two or three had occurred, or when the series was over. The initial convulsions are generally one-sided, affecting the limbs afterwards paralysed, and the later fits have almost always this distribution; sometimes the initial fits are general. In some cases the hemiplegia comes on without any convulsion. In most instances, whether there are convulsions or not, the onset is attended by severe cerebral symptoms, and the child often lies unconscious for several days. Sometimes there is fever and vomiting. In older children there may be some pain in the head, but this symptom, on the whole, is not prominent. In the cases that are secondary to some acute disease, attended with general prostration, or in which such prostration follows general convulsions, the hemiplegia may not be discovered for some days or weeks after the onset. If the paralysis is on the right side, it may be accompanied by distinct aphasia in those children who have already acquired the power of speech. The aphasia passes away completely in the course of a few weeks; rarely it lasts for a month or two.

The further course of the symptoms, and the persistent condition, vary in different cases. In most of them a considerable degree of hemiplegia remains, but it is probable that in some instances a cerebral lesion of the same character is so placed as to cause no persistent symptoms, and there may then be no indication of its existence. I remember, many years ago, finding a large cavity, the size of a hen's egg, in the posterior part of the left parietal lobe, in a man in whom no cerebral lesion was even suspected. With the exception of the half-vision centre, in the posterior extremity of the hemisphere, it is probable that a lesion in early life causes lasting symptoms only when it involves the motor path or motor cortex.

The residual hemiplegia affects the right or left side with about equal frequency. In some cases the amount of the persistent palsy is slight. If at first incomplete, it soon passes away from some part of the side, the arm or the leg. More often it is at first complete, and continues so for a few months, and then power slowly returns in some parts, especially the face and arm, in consequence of the compensation by the other hemisphere. Very rarely the paralysis has been bilateral from the first, and both sides have remained paralysed. This is doubtless the result of a bilateral lesion, as one recorded case proves;* compensation is then impossible, and the palsy remains absolute in the legs as well as in the arms.

The ultimate condition of these cases necessarily depends on the severity of the symptoms. In a large number the arm remains considerably paralysed. Some power is recovered in the shoulder and elbow, and a little in the hand, and as movement returns contracture comes on. In the vast majority the limb becomes the seat of mobile spasm (athetosis, post-hemiplegic chorea), of which these cases present the most typical examples.† There is varying flexor and extensor spasm at the several joints, the variations being greatest in the hand, in which there are often spontaneous movements, quick or slow, while voluntary movement is disordered and ataxic. The condition is that described at p. 85. The subluxation of the middle joint of the fingers, in consequence of the spasm in the interosseal muscles, is generally conspicuous. The active changing spasm is proportioned to the amount of voluntary power; when this is slight there is much fixed spasm, by which the wrist-joint is often strongly flexed. The spasm in the upper part of the arm may be considerable, and may keep the limb rigid, generally in extension. A strong effort with the affected hand causes a corresponding movement in the other hand, and *vice versâ*. In some cases recovery is so great that only a slight amount of inco-ordination remains, and occasionally even this is absent. The paralysed arm is shorter than the other in most of the severe cases, and all the bones, even the scapula, present a diminution in size. The muscles may also be small, but are occasionally hypertrophied from the effect of their continuous over-action. The leg always regains considerable power, and the patient can walk without difficulty, although the growth of the limb may be so hindered as to cause a limp. The knee-jerk is excessive, and a clonus can sometimes be obtained. Spasm is generally slight, but often causes some talipes equinus, or equino-varus, always to be overcome by gentle pressure: the toes are sometimes over-extended in the act of walking. In the face the residual weakness is trifling, but often there is distinct over-action of the muscles on the affected side. This is seen best in the act of smiling, in which a slight movement occurs earlier, and is at first more marked, than on the other side, although a strong movement may be distinctly slighter.

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* Heubner (see p. 461 note).

† On account of the frequency of this symptom the cases have been termed "cerebral spastic paralysis," but the designation is too wide in its meaning to be very appropriate.

As a rule, sensation is perfectly normal on the paralysed side. It is probably impaired in some cases during the early stage, but recovery is constant, so constant that it can only be by the compensation effected by the other hemisphere. In the only case I have seen in which there was any loss of sensation some years after the onset, the loss was clearly functional, hysterical hemianæsthesia; a week later it had disappeared, and existed on the opposite side. Mental defect is very common, and presents every conceivable degree, from a slight amount of hysteria to pronounced iodicy. One of the most severe cases of hysteria that I have seen was in a girl, the subject of old infantile hemiplegia. Another frequent symptom is convulsion, recurring, and resembling idiopathic epilepsy in its course, although not in its origin. Such recurring fits are met with in at least two thirds of the cases. Sometimes they continue from the onset; sometimes an interval of years may elapse before the fits begin, and occasionally the hemiplegia occurs in infancy, and the convulsive attacks are only added when the age of puberty is reached. These cases will be again considered in the chapter on "Epilepsy." The convulsions are almost always one-sided, affecting only the paralysed limbs, and often begin locally after the fashion of "cortical epilepsy." It is, however, a significant fact, indicating how profound is the influence of the repeated discharges on the brain, that the fits may ultimately be preceded by an aura identical with some one of the most common warnings of idiopathic epilepsy, and that minor attacks may be developed which consist only of loss of consciousness.

Pathology.—Very few observations have been made on the condition of the brain in these cases, and those are chiefly on cases long after the onset. The conditions found have varied, and are susceptible of different explanations. In some, a cavity has been found in the central region of the hemisphere, involving the central ganglia, and sometimes extending as far as the cortex of the motor region. If there is an opening on the surface of the brain the condition has been distinguished by a special name, "porencephalia."* The actual cavity may be very large, or may be small, and may appear as if cicatricial contraction had drawn the walls together. On the other hand, there is sometimes no cavity or evidence of extensive destruction of tissue, but some convolutions are small and indurated. In most cases the whole of the hemisphere in which the lesion is found is smaller than the other; the diminution in size may involve all parts of the cortex, and its substance is firmer than normal, and contains more connective tissue.* The condition has been termed "diffuse lobar sclerosis,"† or "chronic encephalitis."‡ Occasionally such a diminution in size of the whole hemisphere is the only visible disease; the atrophy is, however, generally more marked in one part than in another. In the former case it is possible that there has been a primary lesion in one part of the cortex, and the wasting elsewhere has been secondary, although the two can scarcely be distinguished after the lapse of years. We are only considering now the cases in which there was a sudden onset, and it is scarcely conceivable that any disease, affecting primarily in such a manner all parts of a hemisphere, can have come on suddenly, or that actually sudden palsy should be the manifestation of a chronic process. It seems more probable that, in such cases, the diffuse change is really a secondary atrophy.

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* It is curious that in some cases of such cavity there has been a defect in the bone corresponding to the hole in the brain. The origin of such defect in skull and brain is not known, but it is improbable that the condition belongs to the class now under consideration.

Two kinds of lesion have been assumed, in current theory, as the cause of these acute symptoms. According to the one, the primary lesion is, in most cases, vascular occlusion; according to the other, which has been put forward by Strümpell,§ and widely accepted in Germany, it is inflammation of the grey matter of the cortex, "polio-encephalitis," analogous to the inflammation of the grey substance of the spinal cord, "polio-myelitis." Attractive as the latter theory is at first sight, it rests upon no evidence, and, as Sachs has pointed out, the scanty pathological facts we possess are opposed to its validity, as are also the facts of etiology. A primary inflammation of a part of the cortex of the brain is a purely theoretical disease; if it occurs not rarely in childhood (as must be assumed on this theory), pathological evidence of it would certainly have been forthcoming, even more abundant than that of the analogous spinal malady, as every kind of organic brain disease is more often fatal than is disease of the cord. It must be remembered that the general liability of the brain to primary inflammation is very much less than is that of the spinal cord, and we are therefore not justified in relying on analogy as a basis for theory. The circumstances of the onset present a marked contrast to those of the spinal malady, since the latter is scarcely ever secondary to a general disease, while the cerebral lesion, whatever its nature, frequently is.||

The alternative theory, vascular obstruction, has much to support it. It is the common cause of such central cavities as have been

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* Many examples of this are on record. A good instance has been described by Kast, 'Arch. f. Psych.,' Bd. xviii, Heft 2.

† Marie and Jendrassik, 'Arch. de Physiologie,' 1885, No. 1.

‡ Bourneville, 'Recherches sur l'Epilepsie.'

§ 'Deut. med. Wochenschr.,' 1884, No. 44.

|| This paragraph has been left unchanged from the first edition, because the evidence is practically unaltered.

found in some cases. It is a known consequence of such general diseases as frequently precede the cerebral lesion. It has been actually found in some cases of the kind.[*] A cavity in the central ganglia can hardly be due to any other cause than arterial obstruction. But whether this is the result of embolism or thrombosis is an open question. The former has generally been assumed.[†] But in the vast majority of cases of proved embolism there has been an obvious source for the occluding plug, in most cases endocarditis. In some of the conditions in which infantile hemiplegia comes on, endocarditis is most improbable; whereas we know that, at least in the sinuses of the brain, primary thrombosis occurs in children, and sometimes occurs under the circumstances in which infantile hemiplegia comes on.[‡] Hence, while some cases may be due to embolism, it seems on the whole more probable that when the primary lesion is obstruction of an artery, this is the result of thrombosis *in situ*. In the cases in which there is no evidence of softening *en masse*, in which there is no cavity, but only shrinking and induration of part of the cortex, I have suggested that the lesion is probably thrombosis in a surface vein,[§] and that the reason why this lesion is so seldom found post mortem is because in fatal cases the clot usually spreads into a sinus before death, and the case is then regarded as one of sinus-thrombosis. We know that the closure of a vein does not commonly cause softening of the whole of the cerebral tissue from which the blood should pass to the vein, but merely intense congestion, minute extravasations, and punctiform softening, a condition that may well leave the state of atrophy and induration met with in some cases. It is certain that thrombosis may be limited to veins. An instructive case has been published by Money, in which such thrombosis was found after scarlet fever, and the extravasations into the related brain-substance showed that the coagulation had occurred during life.* We must remember that thrombosis in a sinus does not always involve its whole cavity: the clot may be limited to the wall or to one side, and the vessel may remain pervious. In this connection it is instructive to note that in one of my cases the history strongly suggested thrombosis in the superior longitudinal sinus. A child of four months old was prostrated by severe diarrhœa, and the fontanelle depressed; convulsions set in, and the fontanelle became extremely prominent; the child lay motionless for four days, and then, when improvement commenced, hemiplegia was found.

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[*] As, for instance, that recorded by Heubner, 'Med. Wien. Blatt.,' 1883, No. 13. Bilateral palsy and rigidity with trismus developed during prostration after bronchitis in a child one year and four months old. Cavities were found in both hemispheres and the pons, with clots in the middle cerebral arteries, but these clots were "canalised," and the arteries beyond were pervious. Embolism, from ventricular endocarditis, was the assumed lesion, the lining membrane of the left ventricle being thickened. But thrombosis *in situ* could not be excluded.

[†] See Abercrombie, 'Brit. Med. Journal,' 1887, vol. i, p. 1323.

[‡] Heubner assumes that in his case embolism had occurred. There was thickening of the lining membrane of the left ventricle, and he supposed that there had been a ventricular, not valvular, endocarditis, from which the plug had come. This theory, to those familiar with diseases of children, will seem far less probable than that of thrombosis *in situ*, especially when the multiplicity of the cerebral lesion is compared with the fact that elsewhere in the body only one uncertain trace of embolism was found. Moreover the canalisation of a clot formed *in situ* is far more probable than is that of an embolic obstruction.

[§] This has also been accepted as the most probable explanation by Sachs (Clin. Lect. in Volkmann's Series, 1892).

Recurring unilateral convulsions have not quite the same weight as indications of a cortical lesion in these cases as they have in lesions of the brain in adults. They have been met with in several cases in which the lesion was in the central ganglia, even supervening years after the onset.

We cannot yet give any trustworthy explanation of the mechanism of the spasm in the limbs which so constantly accompanies the hemiplegia. It seems to follow lesions of various kinds, degrees, and seat, in the cortex as well as the central ganglia. The far greater frequency (almost constancy) of the symptom after a lesion in early life makes it probable that it is in some way due to the disordered action of centres that remain, and not to the direct effect of the disease itself.

The diagnosis of these cases has to be made chiefly from those in which birth-palsy is one-sided, and this depends on the history of a distinct onset after birth, which is scarcely ever wanting. In cases in which similar symptoms are due to a stationary lesion of chronic character, such as a tumour, the early history is distinctive.

The treatment of the cases in the early stage must depend on the probable nature of the lesion. Whatever be its exact character the treatment for thrombosis in veins and sinuses, described at p. 455, will be most suitable. The after-treatment of the mobile spasm has been considered in the chapter on softening of the brain.

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* Money, 'Treatment of Disease in Children,' p. 445. There had been symptoms similar to these in the cases now under consideration, but the clot did not seem of sufficiently old date to make the case a proof of the relation of the symptoms to the thrombosis.

INFLAMMATION OF THE BRAIN

(ENCEPHALITIS, CEREBRITIS.)

ACUTE INFLAMMATION.

The cerebral tissue, like most other tissues, may be the seat of inflammation, but in no organ has the part played by inflammation in producing morbid changes and symptoms been more variously estimated at different times. This is due to two causes: (1) Inflammation causes softening; hence it was once thought that all forms of softening are inflammatory. It is now known that most are not, but are simply necrotic, due to an arrest of the supply of blood. Nevertheless some forms of local disease, which are probably due to vascular occlusion, are still regarded by some authorities as inflammatory. (2) Inflammation of the surface of the brain accompanies that of the membranes, and it is through the former that many symptoms of meningitis are produced. Hence, although the inflammation of the membranes is the primary condition, and the fact is now recognised in terminology, these cases were formerly called "inflammation of the brain," and are often still thus designated in popular language. These two classes must be therefore put on one side. The residual cases, in which acute inflammation is known to exist, are not numerous. A source of uncertainty regarding the part played by inflammation in the production of local lesions arises from the fact that the ultimate aspect of the damaged region, years after the onset, has alone been observed, and its aspect is not then distinctive. Much weight, however, may be given to the mode of onset; when this is actually sudden, a vascular lesion is indicated, and not a simple inflammation. This point needs attention, even in the acute stage embolic softening has been mistaken for inflammation.

ETIOLOGY.—Acute inflammation of the brain usually results from one of three causes—a traumatic injury, contiguous inflammation, or some septic influence. *Injury* commonly sets up inflammation in the meninges as well as in the brain, but now and then the former escape, and the latter is affected beneath the surface, apparently because the white substance is more easily injured than the grey. All forms of injury may be effective, blows and falls on the head, fractures of the skull, and punctured wounds. It may occur from mere concussion, but is doubtless set up by the mechanism of interstitial laceration. Usually the cerebritis is immediately beneath the seat of the injury; much less commonly it occurs at the opposite side of the brain from *contre-coup*. The latter especially damages the centre of the convolution, so that there results ultimately an irregular shallow depression in its surface. This appearance is often seen near the apex of the temporal lobe in consequence of a blow on the vertex. The inflammation may subside, leaving only the changes above described, or it may go on to suppuration, and the abscess may run an independent course. A punctured wound is very apt to cause an abscess. A very acute form of cerebritis, sometimes involving the whole hemisphere, may follow rapidly on an operation on the brain when aseptic precautions have been imperfect. In other cases of trephining, local inflammation of more chronic course seems to attend the formation of "hernia cerebri." Another frequent cause of cerebritis is *bone disease*, usually actual caries, but sometimes osteitis which has not gone on to caries. The affection of the bone may be traumatic, syphilitic, or the result of contiguous inflammation. The last is almost confined to the bones adjacent to or enclosing the organs of the special senses, the nose, orbit, and especially the ear. The inflammation of the brain thus excited usually accompanies meningitis, but sometimes occurs alone. It usually goes on to suppuration. *New growths* in the brain usually cause softening in the adjacent cerebral tissue, partly necrotic, the result of pressure, partly inflammatory, but the inflammation is slight in degree, is attended with œdema, and scarcely ever presents any tendency to suppuration. In *acute diseases*, especially erysipelas, diphtheria, and typhoid fever, minute foci of encephalitis, characterised especially by leucocytal aggregations, and sometimes by micrococcal infiltration, are often found on microscopical examination. Lesions of considerable size, in acute specific diseases, are generally due to embolism or thrombosis. In simple *vascular obstruction*, the softening which results is chiefly necrotic; on its margin, inflammation occurs, and is often considerable when the obstructing plug comes from a septic source, as in acute endocarditis. Even then, suppuration is extremely rare. In pyæmia, however, the inflammation thus excited always goes on to the formation of pus.

Idiopathic cerebritis is almost unknown. It has been supposed by Strümpell to be the cause of sudden cerebral palsy in children, and the engaging character of this theory of "polio-encephalitis" has led to the use of this name for the affection. But no observations have given distinct confirmation of the view; all are otherwise explicable, and many certainly point to a lesion primarily vascular and not inflammatory. Actual inflammation, however, seems sometimes to affect the nuclei of the ocular muscle nerves, and also those of the bulbar nerves.

Acute functional disturbance of the brain is usually attended by minute changes, and especially by the vascular alterations that always accompany such derangement; these are revealed by the microscope, and may be of such degree as to be distinctly inflammatory. For instance, in hydrophobia the medulla oblongata, the functions of which are so conspicuously deranged, presents such microscopical alterations—aggregations of leucocytes outside the vessels and even in the substance of the nerve-tissue. These changes are not primary, but are secondary to the intense functional disturbance, although they have many of the anatomical characters of inflammation.

PATHOLOGY.—The alteration in the tissue of the brain which results from acute inflammation has been long and accurately known under the name of "red softening," since it is usually, from the first, much redder than the softening which results from necrosis or mere imbibition of effused fluid and is "yellow" or "white." The red tint of the affected area depends partly on the distension of small vessels, but chiefly on minute points of extravasation, and, according to the number of the latter, the colour varies from pale to deep red. The consistence is lessened, from the disintegration of the tissue-elements by effused liquid and the separation of the particles. For the same reason, the affected area is swollen, and, on section, stands up above the level of the adjacent brain-substance. It is never sharply limited, and the consistence of the adjacent tissue is usually lessened. The microscope shows distension of the vessels, especially the capillaries, foci of hæmorrhage, and accumulations of lymphoid (leucocytal) elements in the tissue and around vessels. These are always to be found, but vary in number; when numerous, the softened tissue may have the aspect of actual pus. The proper elements of the tissue—nerve-fibres, ganglion-cells, and neuroglia-cells are in various stages of degeneration, and from all these granule-cells develop. The cell-elements undergo first the stage of cloudy swelling, the nerve-fibres become granular, the axis-cylinder presents fusiform, highly granular enlargements, which, becoming detached, also form granule-cells. At the margin of the inflamed area, the ganglion-cells also pass into the stage of cloudy swelling, but, instead of breaking up, may atrophy or become pigmented, or undergo a sort of vitreous degeneration. These changes in the nerve-elements are the same as are met with in simple necrotic softening, and hence their occurrence does not show that the process was one of primary inflammation.

It is only in the rare cases in which the inflammation is very slight in degree, and there is no actual destruction of nerve-elements, that recovery of the affected part is possible. Small foci of considerable inflammation may ultimately cease to be visible to the unassisted eye—a different thing from actual recovery. In most cases visible damage persists. The complete disintegration of the tissue-elements, and absorption of the remains of vessels, leave a fatty emulsion in the resulting cavity, and to it the remains of blood-pigment (often hæmatoidin crystals) give a yellow or reddish tint, always deeper in the grey than in the white substance. Such a cavity may at last be indistinguishable from one left by a simple necrotic process. Ultimately, the fatty granules may be slowly removed, and a little clear fluid may alone remain. Occasionally a reticulated stroma persists (or is formed) in the softened area; the loculated cavities thus constituted may contract, so that ultimately a sort of fibrous cicatrix remains, with a little adjacent atrophy, the result of the initial œdema around the focus of inflammation. Now and then this cicatrix contains a central nucleus of fatty débris and hæmatoidin crystals. An increase of connective-tissue elements may, perhaps, sometimes cause the development of sclerotic induration in the affected area, but this is far less common than it is in cases of chronic inflammation, venous thrombosis, and compression by meningeal hæmorrhage, and it is doubtful whether such a state ever justifies the diagnosis of an initial acute inflammation. When the process is intense, the lymphoid (pus) cells are so numerous that the softening becomes actually purulent, and a cavity thus results containing pus. This result of inflammation is further described in the chapter on "Cerebral Abscess." A distinctly purulent appearance has been acquired in so short a time as six or seven days. If a very acute and malignant form of inflammation affects an extensive area, rapid and infiltrating suppuration occurs, with acute sloughing of the cerebral tissue.

The traces of inflammation in typhus and typhoid fevers are seldom to be recognised by the unassisted eye. They consist of aggregations of lymphoid cells around the vessels or in the tissue, and may sometimes constitute a mass of relatively large dimensions, comparable in size to a tubercular granulation (Fig. 124). They are not always met with, and are more pronounced in typhus than in typhoid. They have been especially studied by Popoff, who states that the lymphoid corpuscles sometimes enter the protoplasm of the nerve-cells, and may cause multiplication of the nucleus and other changes.*

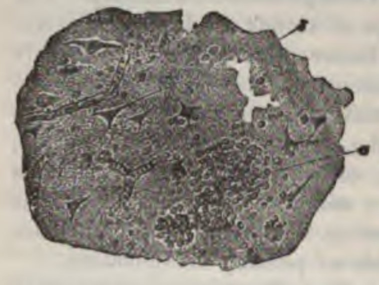

FIG. 124.—Section from the cortex of the frontal lobe in exanthematic typhus: *a*, accumulation of lymphoid elements; *b*, a cavity from which such cells have fallen. (Popoff, 'Virch. Arch.,' Bd. lxxxvii, pl. i.)

The disseminated inflammation that occurs in some cases of erysipelas and diphtheria, and sometimes in septicæmia, is of especial interest on account of the organisms that are associated with it. Sometimes minute points of softening may be visible; more often the changes are only discovered with the microscope. In certain spots micrococci may be seen around the vessels, scattered through the brain tissues, and even densely aggregated in definite "colonies." The condition has been termed "mycosis of the brain."†

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* Popoff, 'Virchow's Archiv,' Bd. lxxxvii.

† See Schüle, 'Virchow's Archiv,' lxvii; Letzerich, ib., Bd. lxxv; and Blaschko, ib., Bd. lxxxii. It must not be confused with "actinomycosis," see p. 467.

Symptoms.—Our knowledge of the exact symptoms of simple cerebritis is meagre, on account of the rarity with which it exists alone. Headache is certainly frequent; vomiting occasional; but optic neuritis is a rare result of the acute process. General convulsions and delirium may also be met with. These symptoms are irrespective of the seat of the inflammation. When the special motor or sensory region is involved, corresponding symptoms may be present, the most frequent being weakness in the limbs on the opposite side, and convulsions, local, or beginning locally. Such symptoms are not uncommon a few days after a cerebral injury, and are often attended by some elevation of temperature. They may pass away completely or may persist in slighter form. They differ from the actual injury to the brain by coming on, not immediately, but two or three days after the injury. The symptoms of an abscess, on the other hand, only develop after some weeks. A young child fell off a chair with a pair of scissors in her hand, and fell on the point of the scissors, one blade of which entered the skull just in front of the middle of the right temporal fossa. It passed in for about an inch and a half in such a direction that the point must have been very near the internal capsule There were no immediate symptoms, but three days after the injury hemiplegia came on; probably the inflammation in the neighbourhood of the wound reached the internal capsule. Sometimes traumatic inflammation seems to set up a degenerative process, which runs an independent course, and is manifested by chronic mental failure.

It is seldom that definite symptoms can be ascribed to the disseminated inflammation that occurs in acute specific diseases. Delirium and somnolence and subsequent mental weakness may be in part due to these changes, but we cannot at present distinguish the effects of the definite lesions and of the influence of the blood-state.

Diagnosis.—In many cases the symptoms are identical with those of meningitis over the corresponding region of the brain. In the form that results from specific blood-states the condition may be suspected if cerebral symptoms become more pronounced after the pyrexia, but our knowledge of the effect of the lesions is still too meagre to permit any definite diagnostic rules to be laid down.

The Prognosis of acute simple inflammation of the brain, as distinguished from that of the membranes, can only be made from the severity of the symptoms. Its Treatment is, on the whole, similar to that of inflammation of the membranes.

Actinomycosis of the Brain.—A few cases are on record in which the peculiar fungus which gives rise to this disease in animals, and sometimes elsewhere in man, has caused foci of inflammation in the substance of the brain. Within these the spherules of the fungus are mingled with pus-cells. Usually secondary, it has been perhaps in one case primary. The symptoms have resembled those of secondary abscess, but the nature of the disease has been suspected only when its existence was recognised elsewhere in the body, and it is too rare to be of practical importance.*

Chronic Inflammation.

Of chronic cerebritis we have also very little exact knowledge. It is true the term "chronic encephalitis" or "meningo-encephalitis" is frequently employed, especially by French writers, but for the most part as a convenient designation for obscure cases, the exact nature of which is unknown. In this loose way the term is applied to all conditions in which traces of inflammation can be found, and the fact of inflammation is allowed to override the question whether the process was primary or secondary.

One class of these cases is that in which the chief alteration is an increase in the connective tissue in some part of the brain, but these cases are described in the chapter on "Sclerosis of the Brain."

The inflammation which surrounds new growths in the brain is often chronic, as well as acute; and acute cerebritis, however caused, may pass into a chronic stage. Indications of extensive primary chronic cerebritis, diffuse in distribution, are sometimes found in cases of diseases usually regarded as degenerative, as general paralysis of the insane. Leucocytal aggregations around the vessels and in the cortex are occasionally met with in this disease, but its extremely chronic course, and the absence of the headache which usually attends encephalitis, discountenance the view that it is primarily inflammatory in its nature. The term "chronic cerebritis" seems, however, strictly applicable to certain rare cases in which a patient suffers from headache and other cerebral symptoms (which may even include optic neuritis),—symptoms that run a chronic course and may end in death. After death the only changes are the microscopical indications of slight general inflammation. For instance, a woman aged thirty-four had attacks of epileptoid nature, some vertiginous, others with transient loss of sight. Some months later she suffered from severe headache, with paroxysmal exacerbations, accompanied by vomiting, intense optic neuritis, slight pyrexia and terminal coma, the duration of the severe symptoms having been about six months. No naked-eye alterations were found in the brain, but slight diffuse inflammatory changes were found throughout its substance on microscopical examination.† A somewhat analogous condition throughout the cortex of one hemisphere has been observed to follow injury in childhood, which led to mental defect and epilepsy, with left-sided weakness and choreoid movements, in a case observed by Fletcher Beach.‡

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* Cases will be found recorded by Keller, 'Brit. Med. Journ.,' 1890; Orlow, 'Deut. med. Wochensch.,' 1890, and Bollinger, 'Munch. med. Wochensch.,' 1887; the last, coincident with an ovarian formation, being believed to be primary and due to unboiled milk.

† Hughlings Jackson, 'Ophth. Hosp. Rep.,' vol. viii, p. 445.

‡ 'Journal of Med. Science,' 1887.

A very rare variety of cerebritis is a disseminated form of chronic inflammation, sclerotic and caseating, due probably to syphilis. A similar affection of the spinal cord is less rare. In the case described by Charcot and Gombault, of which the spinal lesions are shown in Fig. 102, vol. i, p. 337, similar foci of disease existed in the pons, crura, and optic nerves, and are shown in the adjoining figure (Fig. 125). The affected spots were grey in tint, with yellowish centres due to caseation, such as are represented in the smaller figure, which is a section of the diseased area in the pons marked A. Both sixth nerves and the right third nerve are partially degenerated. Thus in the tendency to caseation the morbid process has the characters seen in syphilitic growths, but the histological characters of the diseased areas were those of a sclerotic inflammation rather than of a growth. In these points the process has considerable analogy to that of the more common chronic syphilitic meningitis. The patient had had syphilis nearly twenty years before the onset of the symptoms. Extreme mental dulness existed, and obscured to a considerable extent the symptoms due to the intra-cranial lesions.* Such disseminated inflammation is related, on the other hand, to insular sclerosis (*q. v.*).

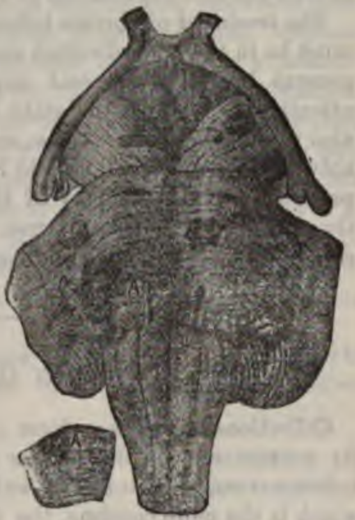

FIG. 125.—Chronic disseminated sclerotic inflammation of pons, &c. (see text). (After Charcot and Gombault.)

It is probable that certain rare forms of chronic softening of the brain (described here as such) are inflammatory in nature, but very little is known of their early stages, and no distinctly inflammatory process can be traced in them at the later period in which they usually come under observation. There is another remarkable group of cases, in which the symptoms suggest chronic focal inflammation, although the nature of the lesion has not, I believe, been ascertained, nor have the cases been hitherto described. The examples I have seen have been in men over forty-five years of age, of gouty diathesis. The symptoms have been those of a chronic focal lesion at the surface of the brain, and have consisted in convulsions beginning locally, hemiplegic weakness, aphasic disturbance, delirium, and sometimes slight pyrexia. They closely resembled in character and course those of a cerebral tumour, but headache has never been prominent, and optic neuritis has always been absent. The course of the affection was marked by successive periods of increase, partial subsidence, and a stationary state. The condition may, however, end in death after a course of one or two years. A chronic relapsing gouty cerebritis affords the best explanation of it.

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* Charcot and Gombault, 'Arch. de Physiologie,' 1873, vol. v, p. 143.

The *treatment* of chronic inflammation, if its existence is suspected, must be in the main directed against its cause. Improvement of the general health is of great importance, together with tranquillity, attention to posture, the state of the bowels, &c. Mercury seems also to influence the process, and, especially in the cases of presumably gouty cerebritis, it should be given by the mouth or inunction in proportion to the intensity of the lesion, the arrest of which sometimes distinctly follows its use. It may also be given in syphilitic cases, followed by iodide and combined with tonics.

ABSCESS OF THE BRAIN.

Collections of pus may form on the surface of the brain or within its substance. In the former case, the membranes, thickened by inflammation, constitute one wall of the abscess. In the latter case, which is the more common, the pus is separated from the surface by a layer of brain-tissue, normal or only slightly damaged, although a communication sometimes exists through this layer between the interior of the abscess and the surface. The white substance of the brain appears to be more prone to suppuration than the grey. An abscess may occur in any part, but is most frequent in the cerebral or cerebellar hemispheres, and is rare in the central ganglia, the pons, medulla, or the middle lobe of the cerebellum. Usually there is only a single abscess, sometimes there are two or more, and occasionally there are many. There is an increasing tendency in modern pathology to ascribe all forms to an organised virus, although the theory is as yet unproved as regards many varieties, especially the deeper traumatic forms.

GENERAL ETIOLOGY.—Abscess of the brain is usually due to injury or to suppurative inflammation, near or distant, from which septic material is conveyed to the brain. The most common cause is disease of the bones of the skull, and of such disease that of the bony investment of the organ of hearing is the most frequent. Injury usually causes an abscess, sometimes directly, but indirectly as a result of bone disease thus produced. We may conveniently divide the immediate causes of cerebral abscess into two classes, the near and the distant. The near causes are the most frequent, and to them no less than 70 per cent. of the cases are due.* Ear disease causes 42·5 per cent., and injury 24 per cent., while disease of the nose, the orbit, non-traumatic caries of other bones, and tumour of the brain give rise to the small remainder. The distant influences cause 15 per cent. of the whole; and the most frequent is suppuration elsewhere than near the bones of the skull, and without general pyæmia. Oidium albicans has twice produced cerebral abscess, but is merely a pathological curiosity. In the remaining 15 per cent. no cause was detected.

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* One hundred and seventy-three of 241 cases collected from various sources, including the 76 cases tabulated by Gull and Sutton ('Reynolds' System of Medicine,' vol. ii, 2nd ed., art. "Abscess of the Brain"). Ear disease, 102 cases; injury, 57 cases.

Age and Sex.—Cerebral abscess is far more common in males than in females, the ratio being as 3 to 1.* The various causes do not, however, influence the two sexes in the same proportion. From ear disease the ratio of males to females is 2 to 1, from injury 5 to 1, from suppuration elsewhere 4 to 1. The greater liability of males to traumatic abscess is readily explained by their more frequent exposure to injury; their greater liability to abscess from other causes is less easy to understand.

No time of life, from birth to old age, is exempt, but the affection is very rare during the first year of life. The following is the relative distribution of 223 cases:

1—9, 24 cases
10—19, 48 „
20—29, 72 „
30—39, 29 cases
40—49, 26 „
50—59, 16 „
60—69, 7 cases
70 and over, 1 case

Thus one third occur in the third decade of life, and one fifth in the second. The twenty years between ten and thirty yield more than half the cases. Those due to ear disease are distributed through life nearly as the total number. Traumatic abscess is relatively more frequent in early life, two fifths occurring under twenty, and nearly one fifth under ten. Pyæmia never, and distant suppuration rarely, causes cerebral abscess under twenty.

GENERAL PATHOLOGY.—Suppuration in the brain, as elsewhere, must be considered as a result of inflammation, and the first stage of the process is apparently "red softening." In this some pus-corpuscles are found in the diseased tissue, and if they are numerous the softened tissue assumes a purulent aspect. It has been thought that pus may be formed without an initial stage of simple inflammatory softening (Huguenin), but the point is doubtful. The pus of a cerebral abscess has a greenish tint, and usually an acid reaction. It is fœtid in one fifth of the cases (Mayer); on what the fœtor depends is uncertain, but it is probably the result of some special septic condition. Under the microscope, the pus-cells in old abscesses are indistinct, having undergone granular disintegration. The wall of the cavity is irregular at first (in some cases for a long time), and there is a tendency to an increase of the abscess by the necrosis of portions of the limiting tissue. The "capsule," which forms after a time, is at first thin and delicate, and gradually increases in firmness and in thickness. It has a smooth inner surface, and its substance is composed of connective-tissue elements, looser externally than in the middle. Beyond it the brain-tissue is often softened by slight œdema, and in the immediate vicinity of the capsule there is some fatty degeneration of the cerebral elements. Doubtless most of the pus-cells are escaped leucocytes, but some may be formed from the cells of the neuroglia. After the capsule is formed, pus-cells still increase in the cavity of the abscess, and, if there is a channel by which the contents may escape, the formation of pus may be continuous and abundant.

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* 174 males to 58 females.

Taking together all cases of cerebral abscess, it is about equally common for a capsule to be present or absent. The time at which one is formed is important, because, if known, its presence affords some indication of the age of the abscess. The question can only be decided by traumatic cases, in which the commencement can be accurately determined. In such cases the first indication of a delicate membrane has been seen at the end of the second week,[*] but it is rarely distinct before the end of the third week,[†] and it only assumes its character as a well-defined membrane with a smooth surface at the end of two months. But an abscess may remain for a much longer time without a capsule. The absence of this is therefore of less significance than its presence. The abscess may be entirely closed, but not unfrequently an opening, sometimes a fistulous channel, connects it with the surface of the brain, especially when there is bone disease, and it may thus communicate with the exterior of the skull. It may also open into the ventricles.

The shape of the encapsuled abscess is more or less rounded, but a recent abscess with adjacent sloughing may be irregular and ill-defined. Rarely it consists only of a fistulous channel extending from the surface of the brain to the ventricles. The size varies in most cases between that of a walnut and a hen's egg, but it may be so large as to occupy apparently two thirds of the cerebral hemisphere, or, on the other hand, it may be no larger than a pea. Multiple pyæmic abscesses are usually small. In about four fifths of the cases the abscess is single—almost always when of traumatic origin, and usually when due to ear disease. On the other hand, when due to distant suppuration, the abscess is single in less than half the cases, and in general pyæmia the abscesses are multiple in two thirds of the cases, and they are generally numerous and small. When multiple, it is about as frequent for there to be two, a few, or many. In half the cases of multiple abscesses, these are situated in the same hemisphere of the cerebrum. Suppuration occurs in both hemispheres only when due to distant septic causes. In one quarter of the cases the abscesses are situated in the same side of both cerebrum and cerebellum. Less frequently there are multiple abscesses in the cerebellum, and the cerebrum is free.

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[*] Lallemand, quoted by Lebert.

[†] Lebert, 'Virchow's Archiv,' Bd. x, 1856, p. 95.

Suppuration occurs in the cerebrum four times as frequently as in e cerebellum, and is very rare in the pons or medulla oblongata.[*] rebral abscess may result from any cause, but cerebellar abscess is oduced almost exclusively by ear disease or distant influences. In he cerebrum, suppuration is a little more frequent on the right side han on the left, but the two hemispheres suffer almost equally from istant causes and from ear disease. The general excess of abscess in he right hemisphere depends entirely on the influence of other local causes than ear disease; these show a remarkable proclivity to affect the right rather than the left hemisphere.[†] Cerebellar abscess, generally due to ear disease or distant causes, corresponds in this respect to the cerebral abscesses of the same origin, and the two cerebellar hemispheres are affected with equal frequency. Suppuration is very rare in the middle lobe of the cerebellum. Any part of the cerebral or cerebellar hemispheres may be affected, but the position of an abscess due to local disease is determined by the situation of its cause.

An abscess, if of any size, exerts pressure on adjacent parts, although not to the same degree as a tumour. The convolutions over it are flattened, and the adjacent brain is anæmic and often softened. Moreover, an abscess in the middle lobe of the cerebellum may cause internal hydrocephalus, just as does a tumour in the same situation.

Anatomical Course.—An abscess which has become encapsuled may remain for a long time stationary. The capsule becomes thick and tough, may even become calcified, and very rarely the contents undergo a similar change.[‡] More often, before a capsule is formed, or while this is still thin, the abscess enlarges, usually more in one direction than in another, and the patient may die from the extensive interference with the cerebral functions. It may ultimately burst into one of the lateral ventricles, less commonly on the surface of the brain. The former occurs in one sixth of all cases; most frequently (one in three and a half) in abscess from distant suppuration (not distinctly pyæmic); less commonly in abscess from ear disease (one in five); still less frequently in traumatic cases (one in nine). The effect of rupture, external or internal, is to excite purulent inflammation, of the meninges in the one case, of the lining membrane of the ventricles in the other, and these cavities become filled with pus. Purulent inflammation of the ventricles may also be excited without actual rupture. Occasionally the ventricles contain an excess of turbid fluid but no pus. An abscess due to bone disease may communicate with the latter by a perforation through the thickened and adherent membranes, and pus may thus be discharged from the interior of the brain through the ear, nose, &c. A very rare accident is hæmorrhage into the sac of an abscess; in one case extensive meningeal hæmorrhage from a vein occurred over an abscess just beneath the surface. Small abscesses are occasionally found in the vicinity of a larger one, and contiguous abscesses may coalesce.

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[*] Cerebrum 186 times (not cases); cerebellum 41; pons 3; medulla oblongata once.

[†] Thus injury caused abscess in the right hemisphere 22, in the left 15 times; disease of the nose, right 7, left 1; disease of the orbit, right 3, left 0; caries of other bones than the temporal, right 4, left 1. The greater influence of these causes on the right hemisphere seems to be too uniform to be accidental.

[‡] Fenman, 'Edin. Med. Journ.,' October, 1879.

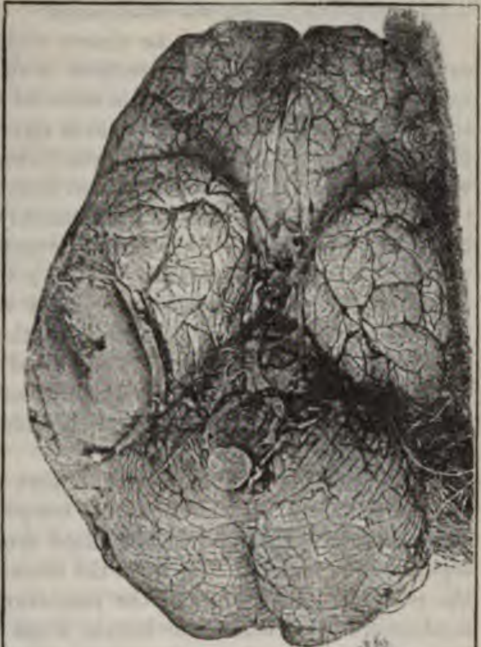
Special Etiology and Pathology.—A. *Local Causes.*—(1) *Injury.*—While the observed proportion of abscesses referred to injury is about one quarter, it is probable that the actual proportion is larger, and that many of the cases in which no cause can be traced are due to some unnoticed or forgotten traumatic influence—a blow or fall on the skull, causing fracture of bone in some cases, or necrosis, although a careful examination has sometimes revealed no injury to the bone. The abscess is usually situated beneath the part struck, near the surface when there is fracture, deeply in the brain when the bone is uninjured. When traumatic necrosis of the bone exists the abscess is sometimes deep, sometimes superficial, and its cavity often communicates with the pus about the bone. In fracture, the inner table is often splintered, spiculæ project into the brain, and the suppuration may be near the surface, or it may be deep-seated and connected by a fistulous passage with the irritating splinter. Another occasional traumatic cause is a penetrating wound, as a stab or a fall on a projecting nail, &c. Traumatic abscesses in the opposite part of the brain are rare; the bone has been fractured or is intact at the part struck. A fall on the occiput has caused an abscess in the frontal lobe, and a fall on the forehead, fracturing the bone, has caused an abscess in the corresponding frontal lobe, and another in the cerebellum.

(2) *Disease of the ear* is the most frequent cause of abscess. The ear disease is usually chronic, and has existed for several years—five, ten, fifteen, and even twenty or twenty-five years—before it caused the abscess; very rarely the mischief has existed only for a few weeks or months. There is usually caries of the bone, following an inflammation of the middle ear or mastoid cells, set up by cold, injury, or more frequently by extension from the throat, sometimes by a polypus in the external meatus. A purulent discharge from the ear (the tympanitic membrane being perforated) has usually existed for a long time. In many cases the abscess has followed the arrest of this discharge; less commonly it has followed an increase in the ear mischief, due to fresh cold or to a blow on the ear.

Occasionally there is suppurative inflammation of the middle ear or mastoid cells, and no bone disease. The tympanic cavity and mastoid cells are separated from the interior of the skull only by a thin layer of bone, which is readily destroyed, and, moreover, this is perforated by small veins which pass from the tympanum to the superior petrosal, and from the mastoid cells to the lateral sinus. Abscess from ear disease is twice as frequent in the cerebrum as in the cerebellum. In the former it is usually in the temporo-sphenoidal lobe, occasionally in the frontal, rarely in the occipital, and still more rarely in the parietal. Abscess of the cerebellum is almost invariably in the hemisphere. In rare cases the abscess is seated in the pons Varolii. Multiple abscesses from ear disease (met with only in 13 per cent. of the cases due to this cause) may be in the same cerebral or cerebellar hemisphere, or in both; they are always on the same side.

The membranes are usually thickened over the diseased bone, but are sometimes normal. Coagula of old date, sometimes breaking down, are occasionally found in the petrosal or lateral sinus. The abscess is rarely superficial; commonly it is seated within the brain, separated from the surface by normal cerebral substance. In other cases an opening exists in this tissue and in the adherent membranes, so that the sac of the abscess may communicate with the diseased bone, and thus with the exterior. In some of these cases it is probable that the abscess commenced in the interior

FIG. 126.



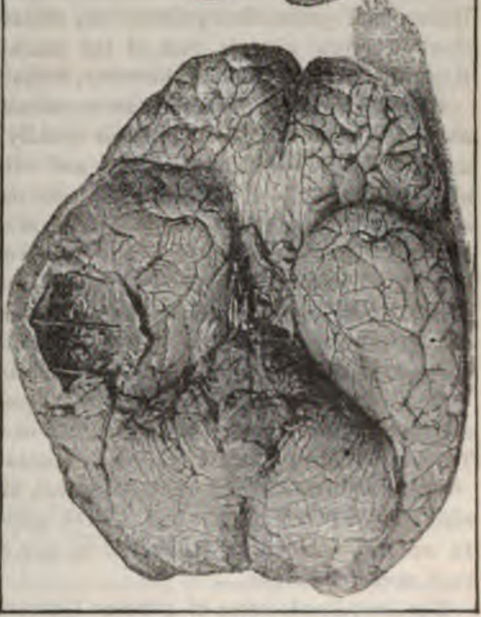


FIG. 127.

FIGS. 126 and 127.—Abscess of right temporal lobe, due to ear disease. The enlargement caused by the abscess is shown in Fig. 126; in Fig. 127 the cavity is opened.

of the brain, and extended outwards towards the diseased bone, until a communication was established.

The mechanism by which an abscess within the brain is produced by ear disease, not directly continuous with the cerebral suppuration, must be by the passage of septic material from the bone disease into the cerebral substance. The arrest or retardation of the circulation in the sinuses may permit this material to reach the brain by the veins which, from ear and brain, pour their blood into the sinuses; but while the current is still flowing this can scarcely occur.* It is not improbable, however, that the perivascular lymphatic canals are the paths by which the infection generally travels; in these inflammation may extend by contiguity, and does so rapidly when septic in nature. The internal carotid artery sends twigs to the interior of the tympanic cavity, and the perivascular sheaths of these have been thought to be the chief channels of infection (Binswanger), but the rarity of abscess in the frontal lobes in consequence of ear disease renders this improbable.

The roof of the tympanum forms part of the middle fossa of the base of the skull, on which lies the temporo-sphenoidal lobe, and the superior petrosal sinus receives blood from both. The bone which separates the mastoid cells from the intra-cranial cavity forms part of the posterior fossa beneath the tentorium, and on it the cerebellar hemisphere lies, while the lateral sinus receives blood from both. Hence, as Toynbee first pointed out, disease of the tympanum causes chiefly cerebral abscess, that of the mastoid cells cerebellar abscess. Exceptions to the rule are, however, occasionally met with.

Chronic disease of the nose is an occasional but rare cause of cerebral abscess (6 cases of 240). There is usually bone disease, involving the nasal, sphenoid, or ethmoid bones, and often syphilitic in origin. In some cases the disease is confined to the nasal mucous membrane, just as the ear disease may be limited to the mucous membrane of the tympanum. The abscess, single in two thirds of the cases, is almost invariably situated in the frontal lobe; in only one recorded case was it in the parietal lobe. Meningitis frequently co-exists. When there is bone disease the sac of the abscess may communicate with this, so that pus is discharged from the interior of the brain through the nose.

Still rarer as a cause of cerebral abscess is *orbital disease* (three cases). In two there was an abscess in the orbit, in one a growth. The suppuration was always in the frontal lobe, and single.

Caries of other bones than temporal, and not due to injury, very rarely causes cerebral abscess. Five cases only were of this nature. In one the caries was syphilitic, in one cancerous. The abscess in each case was single.

The last local cause of abscess (extremely rare) is a tubercular growth in the brain, which has been known to break down into a collection of pus.[*] Other tubercular tumours, and other evidence of tubercle, are usually present, and indicate the origin of the abscess.

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* Adams, 'Glasgow Med. Journ.,' vol. xv, 1881, June, p. 424. The problem of the mechanism is well discussed in this paper.

The Distant Causes of cerebral abscess are morbid processes in some other part of the body by which septic material is produced, and this, passing into the blood-current, doubtless causes the abscess of the brain by septic embolism. The reality of this mechanism has been demonstrated in one case; a cerebral abscess, secondary to suppuration in the lung, was found to contain lung-pigment.[†] Since embolic processes are rarely single, cerebral abscess from distant causes is frequently multiple. From local causes the proportion of cases in which there are more than one abscess is only 13·5 per cent.; from distant causes it is 61 per cent.

In general pyæmia the brain is far less frequently the seat of the secondary suppuration than are some other organs, and it is probable that the brain is less frequently affected in pyæmia due to injury than in pyæmia due to non-traumatic causes. Only 9 of 234 cases of cerebral abscess occurred as part of general pyæmia, and in only one of these was the pyæmia secondary to injury; in one it was post-puerperal; in three it was secondary to abscesses elsewhere, and in two to abscess of the liver produced by dysentery. In one third of the cases the abscess was single, in another third there were from two to five abscesses, in the remaining third numerous small foci of suppuration were scattered through the brain, as many as sixty-eight being counted in one case.[‡]

In an important group of cases (about 10 per cent. of the whole) an abscess of the brain is the result of suppuration elsewhere, generally in the lung, without indications of general pyæmia. This form has been termed "pulmonal cerebral abscess;"[§] the limitation of the secondary suppuration to the brain is remarkable. Pneumonia, imperfectly resolved and breaking down; suppurating cavities in the lungs, left by such pneumonia, or the result of bronchial dilatation; simple fœtid bronchitis; and especially empyema, are the chief causal conditions.[||] It never results from true tubercular cavities. Suppuration in the abdominal cavity is a less common cause, and still rarer is suppuration connected with the limbs. The abscess is single in about half the cases, and is generally situated in the cerebral hemispheres, especially in the posterior lobe. The cerebellum is not often affected from this cause, and never suffers alone. Numerous small foci of suppuration have been met with, but far less frequently than from general pyæmia, which is a less common cause.

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[*] For an instance see D'Espine, 'Rev. méd. de la Suisse Romande,' 1886, p. 371.

[†] Böttcher, 'Petersb. med. Zeitschrift,' 1869, and 'Virchow's Jahresbericht,' 1869, ii, 51.

[‡] It should be noted that pyæmia is not always the cause of a cerebral abscess with which it co-exists. The abscess may be of local origin, and the pyæmia may be the result either of the abscess or of the local cause of the abscess.

[§] Martins, 'Hirnabscesse,' Berlin, 1892.

[||] In diseases within the lung-substance capable of causing it, cerebral abscess appears to occur in about 8 per cent. according to some facts published by R. Nather ('Deutsche Archiv f. klin. Med.,' xxxiv, p. 169). Of ninety-eight cases (forty-nine of gangrene of the lungs, thirty-seven of fœtid bronchitis, and twelve of bronchial dilatation) there was cerebral abscess in eight.

Oidium Albicans.—Strange as it may seem, in two recorded cases* thrush in the mouth was a cause of abscess of the brain. One patient was an infant, the other an adult. An abundant growth of oidium occupied the mouth and throat, and the brain was studded with small abscesses, containing the same fungus. The oidium in the pharynx has been traced into the epithelial layer of the mucous membrane,† and into the interior of the vessels, and it doubtless thus finds its way, with the blood-current, to distant parts.

There remains about a sixth of the total number of cases in which no cause for the abscess could be discovered. In two or three of these congenital heart disease existed, and was regarded as a cause of the abscess, but no mechanism by which the result could be produced has been suggested. In many of these unexplained cases it is highly probable that the abscess was really traumatic, the result of some forgotten fall or blow. In a few cases in which the abscess was situated in the temporo-sphenoidal lobe, it may have been the consequence of disease of the tympanum, overlooked because there was no caries of the bone. It is still an open question whether all cases can be thus explained. and whether there is or is not an idiopathic form of abscess of the brain. If so, it must be ascribed to the localisation within the brain of a specific virus, probably of low intensity, and slow, limited influence. The rare actinomycosis of the brain (p. 467) is, practically, a form of abscess.

SYMPTOMS.—A cerebral abscess originates in inflammation, and constitutes, when developed, a foreign mass within the brain. Ultimately, secondary processes occur, adjacent œdema and inflammation, meningitis, effusion into the ventricles; or the abscess ruptures externally or internally. In some cases of acute and severe disease, the initial suppurative inflammation progresses with rapidity until it destroys life. In other cases it passes into a stationary condition for a time.

The symptoms correspond to the morbid process, being at first those of inflammation, trifling or grave. In acute abscess they are severe, and continue to the end. In the cases of chronic abscess they are sometimes at first so slight as to be overlooked, and may also be trifling when the process has become comparatively stationary, in what is termed the "latent period." Ultimately acute symptoms supervene even in such cases, it may be suddenly, and run a rapid course, to end in death. These constitute the "terminal stage."

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* Zenker, 'Bericht der Gesellsch. f. Nat. u. Heilk.,' Dresden, 1861, p. 62; Ribbert, 'Berl. kl. Wochenschr.,' 1879, p. 617.

† Wagner, 'Jahrb. f. Kinderheilk.,' i, p. 56.

The symptoms that are produced are thus in part those of inflammation; in part they are those of an irritating foreign body, such as a tumour may cause. Their character is determined by the character of the morbid process, and by its seat; but local symptoms, such as afford an indication of the position of the disease, are absent far more frequently than in the case of tumour. This is due to two circumstances. First, abscesses are often situated in parts of the brain, as the temporo-sphenoidal and frontal lobes, in which a lesion often causes no local symptoms, whatever be its nature. Secondly, the pressure of a slowly developed abscess produces less grave effects than does that of a tumour.

Every gradation is met with between the cases of acute abscess with severe cerebral symptoms, running a rapid course, and ending fatally in two or three weeks, and those chronic forms in which the initial disturbance is so slight as to be unnoticed; while the stationary period is marked by complete latency, so that the existence of the disease is unsuspected until the rupture of the abscess, or some acute consequence, causes death. In cases of pyæmia, moreover, the existence of suppuration in the brain may be unsuspected, because its symptoms are masked by those of the general disease.

Early symptoms are most frequent in cases of traumatic abscess, because the initial inflammation is most extensive. When present they resemble those of meningitis, which often co-exists; there is headache, frequently local, vomiting, and febrile disturbance, attended in severe cases by rigors. Convulsions are less common; when general they indicate that the mischief is extensive and severe. Local convulsions occur only when the disease is near or beneath the central convolutions. Paralysis[*] and delirium are rarely early symptoms. In the cases of acute abscess these effects pass on into those presently to be described, such as attend the terminal stage of chronic abscess, and usually consist in delirium, convulsions, and paralysis on the side opposite to the abscess. The temperature is raised, and severe rigors may occur. Delirium gives place to stupor, which deepens into final coma.

These cases of acute abscess are most frequently the result of injury, next of distant suppuration and pyæmic embolism, while they are not common as a result of otitis. The duration of the symptoms is in most cases from ten to thirty days, but sometimes death occurs at the end of a week, and suppuration has been found at the end of thirty-six hours after an injury.[†] A communication between the lateral ventricle and the surface has been established in twelve days.[‡]

The "latent period" of chronic abscess varies much in duration, from two or three months to several years. When, as is often the case, the initial symptoms were slight, its commencement cannot be fixed, but it seems to be occasionally very prolonged; an abscess which has become enclosed in a thick capsule has apparently remained for as much as seven years in one case, and in another for twenty, without exciting symptoms. But the latency is often imperfect; slight symptoms exist, their nature being misunderstood. Headache is the most common, usually not severe. In cases of ear disease it sometimes alternates with otorrhœa. Convulsions may have occurred, and have been thought to be due to idiopathic epilepsy. In a few cases slight mental disturbance, usually melancholic, has been the only symptom.

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[*] Such cortical motor symptoms are met with especially in abscess from lung disease, in which an embolic process occurs in the region of the middle cerebral artery.

[†] Martins, loc. cit.

[‡] Southam, 'Brit. Med. Journ.,' 1892.

The latent period may end suddenly or gradually. The terminal stage is sometimes preceded by a gradual increase in the headache or mental symptoms, by restlessness, irritability of temper, or depression. Rarely, acute symptoms develop and subside, the latency continuing as before. Such an attack is usually due to intercurrent meningitis. As a rule, when the latency is once broken, the active symptoms increase until death occurs.

The characteristic symptoms of abscess, therefore, are those of the terminal stage; these in acute cases are continuous with the initial disturbance, and even in chronic abscess they sometimes exist in slight degree during the latent stage, especially towards its close. Like those of tumour, they may be divided into general and focal, but the former are the more important, and exist alone more frequently than in the case of tumour.

Headache is as frequent as it is in tumour, and presents similar characters. It is more often moderate in degree, but now and then is extremely severe, and has even been known to kill the patient by its violence. It is often increased by posture, and by muscular effort that causes mechanical congestion. Its position corresponds with the seat of the disease, and more frequently than in the case of tumour; but it is sometimes referred to a different part of the head, and the statements made in a subsequent page regarding the pain of tumour are applicable also to that of abscess. In traumatic abscess the pain is generally referred to the situation of the lesion, but in abscess from ear disease pain in the ear often blends with general pain in the head. Occasionally the pain varies in seat from day to day. Once established, it usually persists.

Vomiting is often associated with the headache, and is especially frequent in cerebellar abscess. The same is true of giddiness. Although less frequent in cerebral than in cerebellar abscess, both these symptoms are occasionally very marked in the former.

Optic neuritis is less common in abscess than in tumour, but is certainly more frequent than published facts suggest. It may occur in both acute and chronic abscess, and probably the ophthalmoscope would show that, during the latent stage, it frequently precedes the onset of the more acute symptoms, but it is rather more common in traumatic abscess than in other forms. The aspect of the neuritis is similar to that which results from cerebral tumour.

General convulsions, resembling epileptic fits, are frequent at either the beginning or end of the terminal period. Convulsions which are local in their distribution or commencement, and of cortical significance, are less common, and are usually associated with defective power. Rigidity of the neck, with retraction of the head and slight opisthotonos, occurs chiefly when rupture has excited purulent inflammation about the pons and medulla.

Paralysis, usually hemiplegic in distribution, occurs in about half the cases. It is often slight and very rarely absolute. Sometimes it succeeds a unilateral convulsion. It is considerable only when the disease involves the motor fibres or cortex, and then may affect only the face, or the arm and face.

Sensation is affected much less frequently than motion. When there is hemiplegia there is occasionally diminished sensibility, but this has been a conspicuous symptom only in a few cases in which the internal capsule may have been compressed by an abscess in the optic thalamus, or posterior part of the cerebral hemisphere, or in cases of abscess of the cerebellum compressing the pons. The cranial nerves sometimes suffer, but more often from associated bone disease or meningitis than from the abscess itself. The olfactory nerves suffer only in disease of the nose. The optic nerves are often damaged by optic neuritis, and this may cause loss of sight, but vision is rarely affected by the direct influence of the abscess. The pupils may be unequal, and sometimes irregular, but as a rule they are normal until the terminal coma sets in. The ocular muscles are sometimes involved; there is rarely paralysis of a single nerve, but there may be ptosis, generally on the same side as the abscess. The movement of the eyeballs is sometimes impaired in an irregular manner, when a cerebellar abscess presses on the pons, and there may then be pain in the region of the fifth nerve. Palsy of the facial nerve and deafness are common in abscess from ear disease, but are the result of the caries of the bone. Speech is often slow, but articulation and deglutition are definitely impaired only when the pons is the seat of suppuration or is compressed.

Mental disturbance is among the most frequent of the terminal symptoms—stupor, deepening to coma, and often preceded by delirium, or by conspicuous mental depression or failure, which may terminate the latent stage. When the abscess is seated on the left side, cerebral impairment of speech is occasionally observed.

Of the symptoms outside the nervous system, pyrexia is the most important. It is very frequent during the terminal stage, and sometimes precedes it; while in cases of acute abscess it is present, in greater or less degree, throughout the disease. Rigors often accompany it, followed by sweating, so that the case resembles intermittent fever. The pulse may be frequent, especially when there is fever, but towards the end (and sometimes throughout) it may be infrequent, falling to 50, 40, or 30. A dry brown tongue and constipation are sometimes attended by profound anorexia, and often by vomiting. The sphincters are rarely affected until the final period.

Symptoms according to Seat.—The general symptoms, headache, delirium, terminal coma, optic neuritis, &c., are irrespective of the seat of the abscess, and the chief local symptoms have been already mentioned. It is important to remember that they are seldom present when the abscess is in the pre-central region of the frontal lobe, or in the temporo-sphenoidal lobe, and when an abscess in the central ganglia is small. Otherwise the statements to be made regarding tumour apply also to abscess.

The headache due to a cerebellar abscess is usually occipital, often, in ear disease, darting from the ear to the occiput, and sometimes it is very severe. Vomiting is frequent, and occasionally there is typical cerebellar inco-ordination, probably in consequence of pressure on the middle lobe. The symptoms that may be caused by compression of the pons have been already mentioned. Rigidity of the neck, probably due to slight meningitis, occurs without rupture.

A small abscess in the pons usually causes no symptoms till it bursts, but alternate hemiplegia, or even bilateral paralysis of limbs and face, has been observed, and is common if the abscess is large.

When several abscesses are present, unless they are very small, one is usually much larger than the rest, and determines the symptoms. The minute abscesses which are occasionally scattered through the brain in great numbers in pyæmia seldom cause definite symptoms.

Rupture.—The grave disturbance which marks the terminal period is usually due to the occurrence of inflammatory œdema and softening round the abscess, induced by its extension, and sometimes by some secondary influence, such as a blow on the head, or exposure to cold. Death is sometimes due to the rupture of the abscess, the frequency of which has been already mentioned. External rupture of a cerebral abscess may cause symptoms of violent meningitis, rapidly fatal, but frequently the adhesions of the membranes prevent the escape of the pus on the surface of the brain. A cerebellar abscess more often ruptures into the membranes; and the acute purulent inflammation which results around the pons and medulla usually causes rigidity of the neck, retraction of the head, slight opisthotonos, and even rigidity of the masseters and convulsive shocks.

Rupture into the lateral ventricles occurs in abscesses in all parts of the cerebrum, but is especially frequent in those of the temporo-sphenoidal lobe. The symptoms produced bear considerable resemblance to those of ventricular hæmorrhage, consisting of convulsions and coma; they rapidly deepen to death.

Occasionally a patient with abscess of the brain dies suddenly, and the post-mortem examination fails to reveal the mechanism by which death was produced. A similar event occurs in tumour.

Course.—The varieties which the disease presents in its course have been already mentioned. They may be divided into the following groups, between which, however, intermediate forms occur:

I. Acute abscess, with the early inflammatory symptoms progressing without considerable diminution into a terminal stage, and the whole affection running its course in from one to four weeks.

II. Chronic abscess, in which a latent period intervenes between the early and late cerebral disturbance, and in which the early symptoms are often so slight as to be unnoticed.

(a) The latency is incomplete; some chronic cerebral symptoms occur—headache, convulsions, or mental depression; and these increase and pass gradually into the more severe terminal stage.

(b) The latency is complete, and the existence of any cerebral mischief may be unsuspected until—

1. The terminal stage develops, usually suddenly.

2. Or death occurs from the cause of the abscess, or from some sudden effect of the cerebral lesion. For instance, a boy was struck on the head by a cricket ball; after a day or two he seemed well. Some months later he was found on his face in bed, dead, a sure sign of death from a convulsion. Post mortem an abscess of the brain was found.

3. Or death occurs from some intercurrent disease. Such cases are very rare. The abscess has remained stationary for a long time; the capsule has become thick and even calcified, and the contents have become inspissated. Such cases are the nearest known approximation to recovery from an abscess. But the disease is still there, and its quiescence can hardly be accepted as proof of entire innocuity, since death has resulted from an abscess after a latent period of twenty years.

When nervous symptoms are once developed they usually run a rapid course. Taking all cases in which the duration was noted, the symptoms last less than five days in a fifth, in a third they do not exceed ten days, in about half the cases they do not exceed a fortnight, and in three quarters of the cases they do not exceed a month. On the whole, the symptoms are more often rapid in chronic abscess from ear disease (in which they usually succeed a latent stage) than in traumatic cases (in which there is often no latency). Of traumatic cases, only an eighth lasted less than five days, a quarter did not exceed ten days, and five eighths did not exceed a month. On the other hand, of cases secondary to ear disease, in no less than a quarter the symptoms lasted less than five days, in a third they did not exceed ten days, and in only one eighth did they exceed a month.

Diagnosis.—The extreme variability and frequent latency of the course of cerebral abscess often render its diagnosis difficult and even impossible, and no brain disease of equal gravity so often escapes recognition. The cerebral symptoms are in themselves equivocal, and are common to other diseases, from simple inflammation on the one hand to a tumour on the other. They often derive significance from their association with general symptoms suggesting a suppurative process, especially fever and rigors, but the most important point is their combination with a cause which is known to be capable of giving rise to cerebral abscess, as injury, chronic ear disease, or distant suppuration, especially in the lungs. Such indications are present in about three quarters of the cases. The greatest difficulty is presented by those in which latency is complete, and the terminal symptoms are sudden in onset and rapid in course; the indications of a chronic cerebral disease are then absent, and the terminal symptoms resemble those of apoplexy. If sudden rupture into the ventricles causes merely convulsions, coma, and death, the nature of the case can be suspected only when a cause for abscess exists. It is of great importance, therefore, to make a careful inquiry or search for the slight but significant symptoms that so often precede the terminal attack. In many cases, moreover, an ophthalmoscopic examination would reveal optic neuritis, which is of weight, although not actually conclusive in cases of chronic ear disease, by which it seems sometimes to be produced even without the mechanism of meningitis.

When chronic brain symptoms are pronounced, the chief distinction has to be from tumour. The absence of a cause of abscess is in favour of tumour. Although injury may give rise to either disease, a growth is a much rarer consequence than abscess, is later in development, more uniformly progressive in course, and it more often involves the cranial nerves. The rapid development of severe cerebral symptoms is in favour of abscess, especially if accompanied by considerable fever and by rigors. The diagnosis is of greatest importance when the disease is deeply seated, since an operation would be justified in cases of abscess when it would be impracticable in tumour.

Meningitis and abscess may readily be confounded, and not without reason, since meningitis may cause the terminal symptoms of abscess, and it may result alone from the most frequent causes of abscess, injury and caries of bone. The distinction, therefore, cannot always be absolute. Preceding symptoms are of longer duration in abscess, and meningitis affects the cranial nerves in greater degree, unless the abscess is seated in the pons. The terminal meningitis which results from abscess is usually purulent and due to rupture; it is more rapid in its course than that which exists alone, often begins locally, and preceding cerebral symptoms may materially assist the diagnosis. When these are absent, the diagnosis may be as impossible as in the cases in which there are only terminal symptoms of apoplectic character.

The distinction from simple inflammation of the brain has to be made chiefly in the cases of injury, and then consists less in a distinction between the two than in the recognition of the indications of suppuration over and above those of inflammation. One of these is afforded by the duration of the symptoms. Prolonged traumatic inflammation of the substance of the brain usually results in suppuration, and hence, if severe local symptoms continue for more than a week, the formation of pus is probable. Another is the presence of the general symptoms, especially rigors, that usually attend suppuration. A third is the subsidence of the acuter disturbance, while slighter symptoms persist and subsequently increase.

The difficulty of distinguishing the existence of abscess in a case of chronic ear disease is sometimes very great, because some general cerebral symptoms are occasionally produced by the ear disease, and, as we have seen, these may include distinct optic neuritis. It is, indeed, impossible to lay down any absolute diagnostic rules for these cases. The important fact is that such cerebral symptoms, and especially considerable optic neuritis, are exceedingly rare results of simple ear disease, and in the great majority in which they are found an abscess exists.

The seat of an abscess is inferred, when due to injury, by the position of this, subject to the facts mentioned above, and also to localising indications, which, when present, have the same significance as in cases of tumour. In abscess from ear disease the chief difficulty is the distinction of the temporo-sphenoidal lobe and cerebellum as seats. It depends chiefly on the position of the ear disease, in the middle ear or mastoid cells, and also on the definite symptoms of cerebellar disease, including, especially, pronounced occipital headache, attention to which will often prevent error.

Functional diseases and abscess can only be confounded in the rare cases in which slight general disturbance, such as convulsions or melancholia, attend the latent stage; and the distinction can only be made by the recognition of some other symptom, such as persistent and severe headache or optic neuritis.

Cerebral abscess can only be mistaken for a general disease, febrile or septic, in cases in which such general brain symptoms alone are present as might be produced by a toxic blood state. Usually, however, the co-existence of headache with delirium and stupor, and retardation of the pulse, will prevent this error, if the general relation of the symptoms is carefully watched and duly weighed.

PROGNOSIS.—In a case of abscess, the diagnosis of which is certain, the prognosis is most grave unless the surgeon can give relief. This conclusion is not invalidated by the very long latency occasionally met with, because, in the few cases in which this has continued for several years, symptoms have been completely absent during the stationary period, so that the diagnosis and prognosis of the disease were impossible. Only in the extremely rare cases in which slight but characteristic symptoms subside into absolute latency can the prognosis be at all hopeful. The longer a stationary abscess lasts, the thicker is its capsule, and the less easily is fresh mischief excited, and therefore the better is the prospect. The proved occurrence of calcification of the wall and inspissation of the contents, together with the fact that an abscess may remain latent for as long as twenty years, permits the hope that, in some cases at least, the abscess may not shorten life; and such hope may reasonably be somewhat stronger if the cause of the abscess has ceased to be active, and the patient can be preserved from influences, such as injury, which may again excite the quiescent process. Indications of the rupture of an abscess leave little hope that life can be preserved.

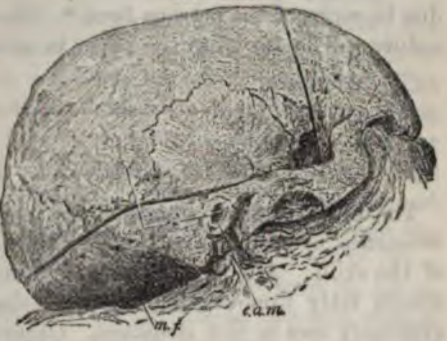
TREATMENT.—Although almost beyond the range of direct treatment, abscess of the brain is not altogether beyond the range of prevention when due to its most frequent cause, local bone disease. This should be subjected to treatment, sedulous and persevering, and, above all, the free discharge of the products of suppuration should be carefully secured, by due and constant attention to the freedom of exit. This remark applies especially to ear disease, in which the first cerebral symptoms have often followed an arrest of the discharge. These measures should be continued even when there is reason to fear that suppuration in the brain has already occurred, for the unfavorable course of cerebral abscess has been certainly accelerated by subsequent increase in the inflammatory and septic mischief in the ear.

The improvement of the health of the patient is of the greatest importance. Tonics, cod-liver oil, and fresh air tend thus to lessen the tendency of the cerebral disease to spread. Of especial importance also is the avoidance of exposure to cold, and of blows or falls on the head. Each of these causes, in several recorded instances, has distinctly excited the disease to fresh progress. If the symptoms should fortunately become quiet, and complete latency be developed, the same care must be maintained for years.

Acute cerebral symptoms must be treated in the same way as those that are due to simple inflammation. Rest, cold to the head, sometimes counter-irritation, as by a blister to the occiput, are the most important measures. The treatment of special symptoms must be conducted on the same principles as in the case of cerebral tumour.

Surgery has endeavoured, and with success, to afford the direct relief which medicine is unable to give. The pus has been evacuated by means of trephining, and the patients have recovered, provided antiseptic precautions were carefully observed, and the cavity, if necessary, drained for a time. But, until recently, success has been obtained almost exclusively in cases of traumatic abscess. In these the abscess is seated, as a rule, beneath the place of injury, and the guidance thus afforded enables the operation, as a rule, to be correctly localised. For instance, a boy struck his forehead and suffered afterwards from headache and retching. At the end of seven weeks hemiplegia came on, with double optic neuritis. The frontal bone was trephined; the dura mater beneath was healthy, but, an aspirator needle being plunged into the brain, three drachms of pus escaped. The boy recovered, but with loss of sight from the optic neuritis.* This case illustrates forcibly a rule which is also indicated by the facts of many unsuccessful cases. If pus is not found on the surface of the brain a fine trocar should be plunged into the cerebral substance. In more than one case of unsuccessful trephining, the post-mortem showed that this procedure would have reached the abscess. But sometimes, when such an abscess is some distance from the surface, every effort to find the pus may fail. Occasionally the operation has merely aided a process which nature was endeavouring to effect. For instance, a girl, aged eight, fell and cut her forehead. The wound healed quickly, but four months afterwards an abscess formed over the bone, and opened spontaneously. A probe passed through carious bone into the interior of the skull. The trephine was applied, a wine-glassful of pus escaped, and the patient recovered.† In any other than traumatic cases it is seldom that the symptoms are so localised as to indicate with precision the seat of the abscess, but when they are an operation has been sometimes successful, since their significance has been better understood during the last few years. The evacuation of an abscess causing cortical convulsions has been followed by permanent arrest of the latter, without excision of the discharging centres.

FIG. 128.



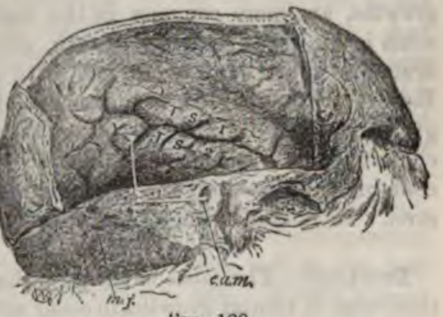


FIG. 129.

FIGS. 128 and 129.—Dissections showing the guide adopted by Mr. Barker in successful trephining for abscess from ear disease.

Until recently no case of abscess from ear disease had been cured by trephining, because the guidance of localising symptoms is, as a rule, absent on account of the position of the abscess. The practicability of the surgical treatment of these cases has now, however, been proved by several successful operations, the surgeon being guided by the most frequent seat of suppuration produced by such disease as the patient presents. One of the earliest of these was that of a patient under my care at University College Hospital, in whom Mr. Barker trephined over the temporo-sphenoidal lobe, in which an abscess was supposed to be, and the trocar, on its first introduction, reached the pus; the abscess was emptied, and the patient made a good and permanent recovery. The cerebral symptoms were slight, as in most cases of abscess in this situation, and consisted only in inequality of the pupil, vomiting, and optic neuritis of considerable intensity and very rapid development. With these symptoms the patient had had several rigors. The precise seat for the trephining was determined by Mr. Barker by means of a careful dissection, represented in Figs. 128 and 129, and was at a point one inch and a quarter behind the external auditory meatus, and the same distance above its level. This point is over the second temporal convolution, and a trocar plunged in at this spot and directed forwards will probably enter an abscess due to caries of the petrous bone.[*] The details of the surgical procedure will be found in the paper in which this case is related.[†]

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* Hulke, 'Medico-Chirurgical Trans.,' vol. lxii. 1879.

† Watson, ib., 1870, p. 353.

INTRA-CRANIAL TUMOURS.

The brain is often damaged by new growths which arise in its substance, or spring from the enclosing membranes or from the bone of the skull. Almost all forms of tumour are met with, but some—simple fatty tumours, for instance—that are common elsewhere are extremely rare in this situation. Others, such as massive tubercular growths, are more common in the brain than in other parts. Sarcoma is not rare; and one growth, glioma, is almost confined to the brain and spinal cord, being met with elsewhere only in the retina. Besides these, the list of intra-cranial tumours includes syphilitic growths, which are also common, cancer, fibroid and bony tumours, cholesteatoma, vascular or erectile growths, psammomata or tumours containing brain-sand, and parasitic tumours, echinococci and cysticerci. Intra-cranial aneurisms are considered in a separate chapter.

Etiology.—The cause of most of these tumours is not less obscure than that of similar growths in other situations. But the comparison of a series of cases brings out certain general etiological facts. The liability of males to suffer is twice as great as that of females. Of 650 cases of various forms of intra-cranial tumour, 440 occurred in males, 210 in females.* The greater liability of males is not due, as might be imagined, to the influence of syphilis, for it is true (as will be seen) of all forms of tumour, with the apparent exception of sarcoma. The difference has been ascribed to the influence of traumatic causes, but it is not probable that this accounts for more than a small part of the excess, since it is rarely to be traced, and the difference in the sexual liability is as marked in the case of children as in that of adults.

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[*] Dean has pointed out that by trephining the same distance behind the meatus, and only a quarter of an inch above its level, the lateral sinus is exposed, and by extending the opening, the temporo-sphenoidal lobe above, and the cerebellum below, can be explored with the trocar—a rule that may be useful when it is doubtful in which structure an abscess is situated; it enabled him to empty successfully a cerebellar abscess ('Lancet,' 1892).

[†] 'British Medical Journal,' 1886, ii, Dec. 11th, p. 1154. In a preceding case, trephined successfully by Schede, a fistulous track led from the surface to the abscess.

This difference is not, however, equally marked in all forms of tumour. It is greatest in the case of tubercle and of glioma. Of the tubercular tumours and of gliomata, nearly two thirds of each were in males, and the same proportion of cases of cancer. On the other hand, of 56 cases of sarcoma 26 were in males and 25 in females.

No time of life is exempt, unless it be the first six months; but cerebral tumours are not common in old age. The largest number of cases occur in childhood and in the active period of adult life. The first twenty years furnish one third of the cases, the second two fifths, and the third one fifth. The proportion in the first decade, 18·5 per cent., falls to 14 in the second, and rises to a maximum of 20 in the third. In the fourth it is the same as in the first, 18·5 per cent., and it falls to 14 per cent. in the fifth, while after fifty years of age the cases rapidly become less numerous. The frequency in early life is largely due to the fact that at this period tubercular growths are so common; if these are excluded, the proportion in the first twenty years of life falls to one fifth, while that in the second rises to one half, and that in the third to one quarter of the total number. Age has little influence in relation to sex. The relative affection of males and females is nearly the same in each period of life, but there is a marked tendency, as in so many diseases, for the sexual difference of the early and adult periods to disappear in the decline of life; over fifty the two sexes suffer equally.

Tubercular tumours of the brain occur at all ages up to seventy, but, as just mentioned, they are most frequent in early life. Three quarters of the cases occur during the first twenty years, and in one half of the whole the patients are under ten years of age. Glioma is most common during active adult life; one half of the cases occur between twenty and forty, one quarter between forty and sixty, and one fifth during the first twenty years. The relative distribution of the cases of sarcoma is nearly the same as that of glioma. Parasitic tumours are most common between ten and twenty, next between twenty and thirty, while under ten and over thirty they are very rare. The youngest sufferer was aged six, the oldest sixty-six. Tumours diagnosed as carcinoma have been met with at all ages, but one half were in patients between forty and sixty years of age, and only two in the first twenty years of life. The other forms of tumour are too rare to permit their relation to age to be stated, further than the fact that none of them seem to have been met with hitherto in patients under twenty.

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* This, and the other statistical conclusions given in the text, have been obtained by a combination of the cases contained in the collections made by Ladame ('Hirngeschwulste,' Würzburg, 1865) and Bernhardt ('Hirngeschwulste,' Berlin, 1881). They agree, in the main, with the valuable collection of facts made by Dr. Allen Starr,

Two forms of cerebral tumour, tubercular and syphilitic growths, depend upon diathetic influences. In most cases of tubercular tumours there is a family history of phthisis; the patients are frequently ill-nourished, and if adults, often present evidence of chronic lung disease. Syphilitic growths, although not unknown in the inherited form, occur chiefly in the acquired disease. The period after infection at which they develop is especially from the fifth to the twelfth year, but they have been met with as early as twelve months and as late as fifteen years after the primary sore.

Little is known of the influences which determine the occurrence of other forms of intra-cranial tumour. It is rare for any indication to be obtained of an inherited tendency to morbid growths elsewhere. There is little doubt, however, that traumatic influences, falls and blows on the head, are occasionally the immediate excitants of a growth, since the symptoms have been observed to follow a blow, and the tumour is found to correspond in position to the seat of the injury. Such a relation has been observed in almost all forms of tumour, especially in syphilitic and tubercular growths, and in sarcomata of the dura mater. Traces of the traumatic mischief may or may not be visible after death, and apparently the nutritive changes consequent on a mere concussion may be the starting-point of a tumour. In some cases local symptoms of irritation, which were continuous with those of a subsequent tumour, commenced two or three days after the injury, and must have been due to inflammation, by which the growth was presumably started. But the cases in which a traumatic cause can be traced form a very small proportion of the total number, and the extent of this influence may readily be overrated. It should be remembered that a fall may evoke symptoms of an early growth which existed before the accident.

PATHOLOGY.—Various classifications of intra-cranial growths have been proposed, founded, for the most part, on the structures in which they originate, but such classifications are of small practical value. It is more convenient to describe the various growths in the order of their frequency. If they are grouped, as an aid to memory, they may be placed in seven categories.

I. Diathetic:—Tubercular and syphilitic.
II. Sarcomatous:—Glioma, sarcoma, myxoma.
III. Carcinoma.

IV. Osteofibroid :—Fibroma, osteoma, osteofibroma.

V. Miscellaneous :—Cholesteatoma, lipoma, vascular or erectile tumours, psammoma, neuroma.

VI. Parasitic :—Echinococcus and cysticercus.

VII. Simple cysts, either connected with the choroid plexus, or very rarely in the substance of the brain.

Excluding syphilitic growths (the frequency of which is not fairly represented in published lists of fatal cerebral tumours), tubercular growths constitute more than half the total number of cases, and gliomata and sarcomata together about a third, glioma being rather more frequent than sarcoma. Thus the two groups, tubercular and sarcomatous, constitute together about four fifths of non-syphilitic tumours of the brain. Carcinoma is much less common, but its actual frequency is uncertain, since many cases of glioma and of sarcoma have certainly been recorded as cancer by older writers. The statement sometimes made, that cancer is one of the most common forms of cerebral tumour, is entirely unsupported by authenticated facts. According to published cases, parasitic tumours are nearly as frequent as carcinoma, but their actual frequency doubtless varies much in different localities, and they are more likely to be published than are cases of cancer. The other forms of tumour are rare; all that can be said is that fibroma, cholesteatoma, and osteoid tumours are more common than vascular tumours (angioma), psammoma, lipoma, or neuroma, the last two being the most rare of all the tumours of the brain.

The relative frequency with which tumours are situated in different parts of the brain is as follows :—Cerebral hemispheres (excluding central ganglia), 297; cerebellum, 179; pons, 59; central ganglia, 48; medulla oblongata, 31; corpora quadrigemina, 13; crus cerebri, 10. Thus the cerebral hemispheres are the most frequent seat of new growths, but when the smaller bulk of the cerebellum is considered, its tissue is evidently far more prone to give rise to neoplasms than is that of the cerebrum.

If we take all forms of intra-cranial tumour, the relative frequency with which the lesion affects the different parts of the brain is as follows. Of 718 separate growths (several existing in some instances), the order of damage was—cerebral hemispheres (white substance and cortex), 297; cerebellum, 179; base of brain, 76; pons, 59; central ganglia, 48; medulla oblongata, 31; corpora quadrigemina, 13; crus, 10. Growths are also occasionally met with in the pineal gland, pituitary body, and the choroid plexus.

Tubercular tumours of the brain occur in the form of solid, firm, rounded masses, having little resemblance to tubercle in its miliary form. They commonly vary in size from that of a pea to that of a walnut, and occasionally attain still larger dimensions, that of a hen's egg or even of the closed fist. Their section presents a uniform, opaque, cheesy aspect, sometimes softened here and there, but never in a large area. The periphery of the mass is softer, greyish, and translucent, and the adjacent brain tissue may be softened. Hence they are readily detached. It is in this grey zone that the growth of the tumour occurs by the development and coalescence of miliary granulations, which quickly degenerate and blend with the caseous mass. The growth apparently takes place in the lymphatic sheaths of vessels, and within the vessels thrombosis may occur. Hence the absence of vessels within the mass, and the quick and uniform degeneration of its elements. If such a tumour ceases to grow, the peripheral layer may undergo a fibroid change so as to form a capsule. Occasionally the degenerated mass undergoes partial calcification. Rarely the tissue softens, and a collection of pus results, increased perhaps by adjacent inflammation. Usually miliary tubercle is found elsewhere in the body, sometimes in the meninges. Occasionally these growths (like bone disease) may be the sole lesion, and hence they are sometimes called "scrofulous" tumours, but of their tubercular nature there is no doubt. They compress the brain tissue, which atrophies before the growth (Fig. 130); they do not infiltrate like some other tumours. They generally occur within the cerebral substance, without apparent connection with the membranes; it has, indeed, been maintained that they are always connected with and spring from a fold of the pia mater, but this cannot be distinctly traced in many cases. When such a tumour reaches the pia mater, this is thickened and often adherent to the dura mater. Now and then tubercular tumours spring from the dura mater, and merely compress the brain without invading it.

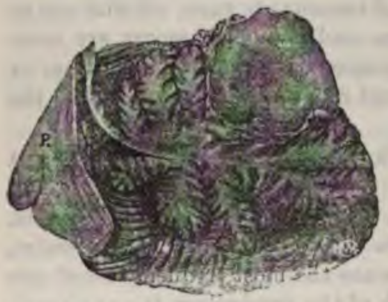


FIG. 130.—Tubercular tumour of the middle lobe of the cerebellum. The patient, a young child, was admitted into University College Hospital, dying from general tuberculosis. P. Pons.

The most frequent seat of such growths is the cerebellum (in which one third occur), either the hemisphere or, as in Fig. 130, the middle lobe, next (and almost as frequent) the cerebrum; while the other parts are affected less frequently and in the following order: pons, central ganglia, crura cerebri, medulla oblongata, and corpora quadrigemina. There is frequently more than one tumour. Of 183 cases, in 83 there was only one tumour, in 100 there were more than one. It is equally frequent for two or more than two to exist, and there may be many—ten, twelve, or even twenty, of various sizes. When there are more than one it is equally frequent for the tumours to occupy the same or different parts of the brain (the white substance and cortex of the cerebral hemispheres being considered as one part). In very rare cases there has been no mass of tubercle, but the whole brain was infiltrated with minute miliary concretions, none exceeding a barleycorn in size (Gee, Baly).

Syphilitic growths (syphilomata, gummata) are certainly more common than is suggested by the frequency with which they are found post mortem, because they are more amenable to treatment than any form of tumour. They usually vary in size from that of a hazel-nut to that of a chestnut; in shape they are somewhat irregular and nodular. Their consistence is not uniform; they are firm, but not equally so in all parts. The section presents an irregular caseation; cheesy spots, isolated or coalescing into irregular tracts, are separated by a firm grey or reddish-grey fibrous tissue. The growing surface is usually grey and gelatinous. The adjacent brain-substance is softened and more or less displaced, but is not infiltrated by the growth. The more irregular surface and the irregular caseation are distinctions from tubercular tumours. Sometimes these tumours are met with in a state of more advanced degeneration, probably under the influence of treatment; they are shrunken, very hard, and fibroid, and may be surrounded by an indurated capsule. The process of obsolescence may go so far that only an indurated cicatrix is left. Syphilomata are rarely met with in the cerebellum or the corpus striatum, but are commonly situated in the cerebral hemispheres or the pons. They are usually superficial in position and attached to the pia mater; even when apparently more deeply seated a connection may usually be traced with a fold of pia mater between two convolutions. The dura mater is often adherent to the growth, which sometimes has obviously commenced in this membrane and thence invaded the brain. In such a case the invasion may be clearly observed to take place along the walls of vessels, probably in the perivascular sheaths. Hence it is probable that in the very rare cases in which no connection with membranes can be traced, the growth proceeds from the vessels. Although these tumours are rarely met with in the central ganglia, they occasionally occur beneath the optic thalamus, as an ingrowth from the neighbourhood of the crus. When the growth is superficial, the membranes in the vicinity often present the indication of chronic inflammation with much thickening.

The Sarcomatous group comprehends a variety of tumours differing much in structure, but all connected by intermediate forms. They consist of round, oval, or spindle cells, varying in delicacy, and with more or less intercellular and fibroid tissue. There is, however, an important difference in their mode of growth. Some infiltrate without displacing the cerebral substance, and have no sharp outline. Others displace rather than invade the brain, have a well-marked outline, and are surrounded by a zone of softening in consequence of the destruction of the nerve-elements before the morbid growth. The former, from their infiltrating character, must grow in the neuroglia, and arise by a modification of its elements. Hence they are termed *gliomata*, and the tumours, before which the brain tissue undergoes destruction or displacement, are alone to be regarded as true *sarcomata*.

The former are composed of more delicate, the latter of less delicate tissue elements.

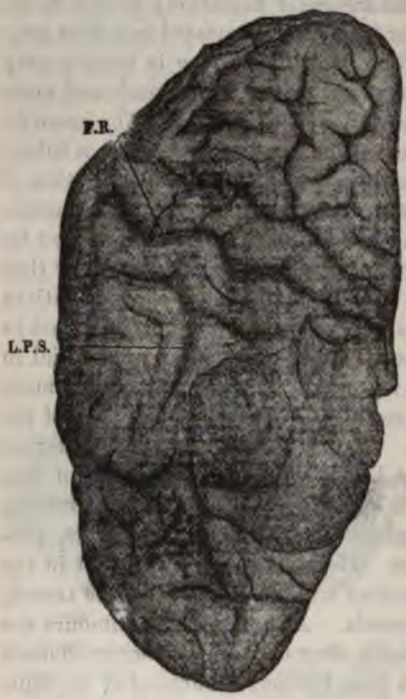
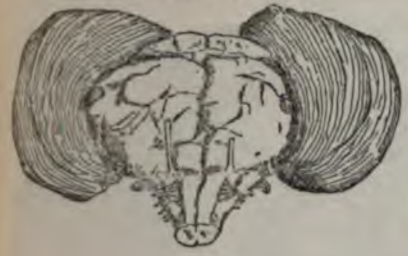


FIG. 131.—Infiltrating glioma of the cortex of the left superior parietal lobe.

Gliomata.—The infiltrating character of gliomata was not that on which their designation was primarily based. Their elements were supposed to resemble those of the neuroglia; but the resemblance is never close, and they rarely present the stellate cells which characterise the normal interstitial tissue of the brain. Their elements vary as much as do those of other forms of sarcoma; sometimes round, oval, or fusiform cells predominate or may alone be visible. But the cells are always delicate; their outlines are difficult to recognise, and they are easily destroyed; so that their round or oval nuclei may alone be visible in a scraping. The interstitial tissue varies in amount; it is usually very delicate and homogeneous, but sometimes is so abundant that the tumour has a mucoid character (myxo-glioma), rarely fibrous (fibro-glioma). Their tint is grey or reddish grey, and often closely resembles that of the brain tissue, being a little paler than the grey matter of the cortex, and their consistence is nearly that of the cerebral substance. The rare fibroid form is firmer, the mucoid form softer than the brain. Moreover they are prone to undergo softening, which may be so considerable and extensive as to convert the growth into a cyst, the wall composed of new growth, and containing, in its cavity, only the débris of its cells. The tint of the tumour depends on the varying amount of vessels which it contains. In soft tumours the vessels easily rupture and give rise to hæmorrhage, so that extravasation may infiltrate a large part of the growth (as in Fig. 133), or an extensive hæmorrhage may occupy a cavity within it. It is rare, however, for the hæmorrhage to escape beyond the limit of the growth. From the infiltrating character of a glioma, it usually causes simple enlargement of the part of the brain in which it occurs; adjacent parts may be compressed by the enlargement, and may be displaced to some extent (as in Fig. 135), but there is not such simple displacement as by non-infiltrating growths. When the pons is the seat of the new growth, the whole of the pons may be greatly enlarged; the basilar artery may restrain the swelling, and ultimately be concealed in the bottom of a deep fissure between the two parts, as in Fig. 134. When a glioma reaches the surface, outgrowths, sometimes pedunculated, often form. Sometimes small separate growths are met with on the surface, as in Fig. 135. It is rare for adhesions to the dura mater to occur. The degree and rapidity of the destruction of the nerve-elements by the infiltration varies much in different cases. Sometimes nerve-fibres persist, and retain their conducting power even in a part which is almost entirely infiltrated with the new growth. As already stated, there is no sharp boundary to the tumour, and it is only in the white substance of the brain that the difference in tint enables its limit to be recognised by the naked eye. In consequence of these characters, curious errors in pathological diagnosis sometimes occur. Cases of uniform infiltration of the pons with a myxoid glioma have been described as "hypertrophy of the pons."* In cases in which the growth has broken down, or has been the seat of hæmorrhage, the existence of the tumour may readily be overlooked, and such cases have been described as simple softening

FIG. 132.



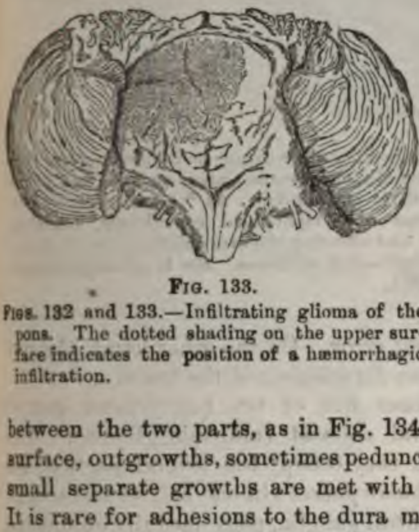


FIG. 133.

FIGS. 132 and 133.—Infiltrating glioma of the pons. The dotted shading on the upper surface indicates the position of a hæmorrhagic infiltration.

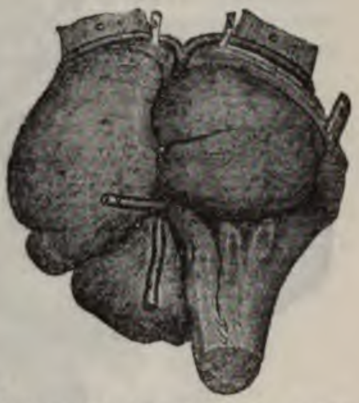


FIG. 134.—Glioma of the pons. V,V, vertebral arteries; the basilar is entirely concealed in the deep groove between the projecting growth on each side. On the right an extension downwards has compressed the right side of the medulla; C, C, crura cerebri; 3, 3, third, and 5, fifth nerve. The patient was a boy, aged eight, who presented during life paralysis of the right fifth, sixth, and seventh nerves, and of the left arm and leg.

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* It is certain that all the alleged examples of hypertrophy of the pons are cases of infiltrating glioma (see Money, 'Med.-Chir. Trans.,' vol. lxvi, 1883).

or simple hæmorrhage. Most of the cases in which optic neuritis has been described as accompanying hæmorrhage or softening have been cases of glioma. A microscopical examination of the margin of the softened part will always prevent error. On the other hand, the rare fibroid forms have been mistaken for sclerosis of the brain.

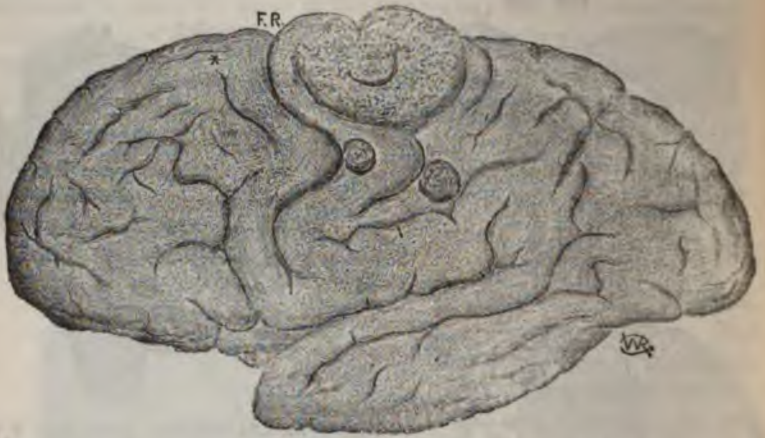

FIG. 135.—Tumours, gliomata of left hemisphere, a large one infiltrating the superior parietal lobule, two smaller growths, one pedunculated, on and just behind the middle of the ascending parietal convolution. At * another growth lay beneath the cortex. The symptoms were right-sided convulsions, beginning with pain in the elbow or shoulder, and affecting the arm chiefly, a few limited to the leg. There was also general right-sided weakness. See Hughlings Jackson, 'Med. Times and Gaz,' 1875, i, p. 661.

Gliomata are single nine times out of ten (sixty-three out of seventy cases). Rarely several co-exist (multiple glioma). The order of frequency with which they occupy the several parts of the brain is as follows:—cerebral hemispheres (in one half of the cases), cerebellum (in one quarter), central ganglia, pons, medulla oblongata, crus cerebri, corpora quadrigemina.

Sarcomata may occur within the substance of the brain, or may spring from the membranes, pia mater or dura mater, or from the bone, especially at the base. When they commence in the bone they may perforate the dura mater, but in this case, and also when they commence in the dura mater, they do not usually invade the pia mater, but merely compress the adjacent part of the brain, sometimes causing a depression of considerable size. Within the cerebral substance they do not infiltrate like gliomata, but have a well-marked limit, beyond which the brain tissue is usually softened, so that the tumour can be easily detached. In each situation they may be hard or soft in consistence; the softer forms are often very vascular, and constitute one form of "fungus hæmatodes." Such tumours, growing from the dura mater or bone, may perforate the skull and be felt as swellings on the surface, softer than nodes, for which they are sometimes mistaken. Occasionally these growths have a spongy section. Those which grow from the dura mater over the convexity often attain a large size; I have seen one that had the dimensions of a turkey's egg. Those which spring from the membranes at the base are usually small, firm, and more or less rounded. The softer forms may consist chiefly of round cells, but the firmer varieties are composed of spindle-cells, varying in size, less delicate than those of glioma, and often separated by tracts of fibrous

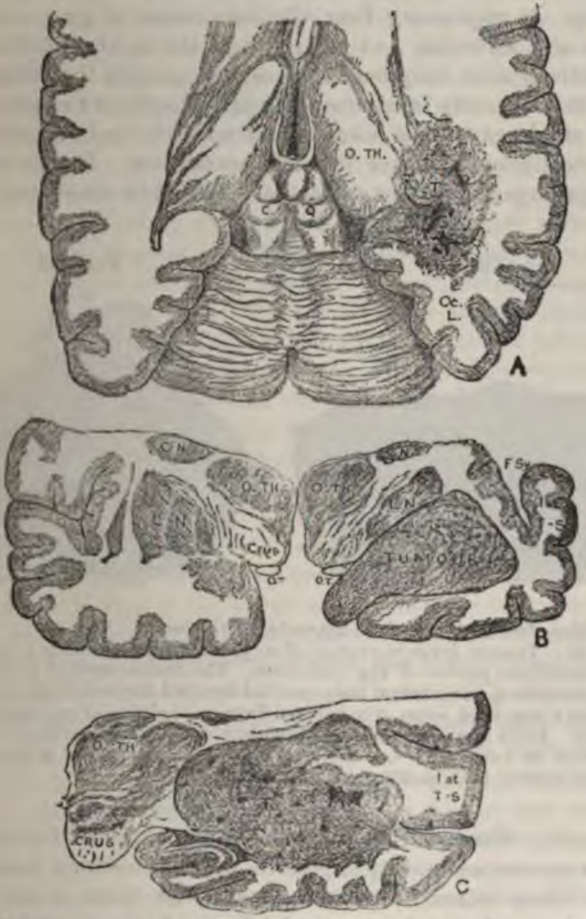

FIG. 136.—Tumour, beginning between the right optic thalamus and first temporal convolution and extending inwards from the temporal lobe, so as to compress the optic tract (Fig. 71, p. 145). A, horizontal section through the posterior parts of both hemispheres; T, tumour, with small irregular cavities, due to softening of its tissues; B, transverse section through the middle of the thalamus; C, through the posterior part of the thalamus. The patient suffered from hemianopia, fits beginning with an auditory aura (sound of bells referred to the opposite ear), optic neuritis, and hemiplegia.

tissue. Occasionally the spindle-cells are arranged in concentric nests.[*] They may contain nucleated cells, such as are so abundant in the so-called "myeloid" tumours. In the substance of the brain these growths are usually single, but occasionally more than one exists; when they spring from the dura mater or the base they are often multiple. They are more frequent outside than within the brain, but may occur in any position, and are seated in the central ganglia more frequently than gliomata.

Carcinoma of the brain, almost always soft, may be primary or secondary. It may spring from the dura mater or grow within the brain in any situation, but especially in the cerebral hemispheres. It is relatively more frequent in the central ganglia than other forms of tumour. It partly infiltrates and partly displaces the brain tissue. Usually single, carcinoma is sometimes multiple, and now and then a symmetrical growth occurs in each hemisphere. In one case, for instance, a large secondary tumour was situated in the central ganglia on each side. Cerebral cancer is usually vascular, and forms sharply limited tumours, containing large cells which sometimes have several nuclei. When seated in the dura mater the tumours are usually small and vascular, and may perforate the bone, or grow into the brain, and, like the similar form of sarcoma, are often termed "fungus hæmatodes."

FIG. 137.

FIG. 138.

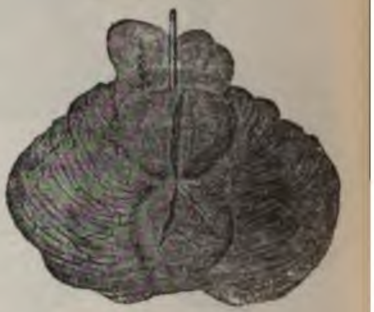

FIG. 137.—Cancerous growth in the cortex of the brain.

FIG 138.—Tumour (cysto-myxoma) of corp. quadrigemina with a cyst in the superior vermiform process of the cerebellum. The tumour seemed to have sprung from the posterior quadrigeminal body, and had involved the medullary velum. The patient was a boy, aged seven, who suffered from unsteadiness of gait and divergent strabismus. Acute symptoms, occipital headache and vomiting, with a tendency to fall forwards or backwards, and optic neuritis supervened after a fall. (Kohts, 'Virchow's Archiv,' lxvii, p. 425, and pl. xiv.)

The rarer forms of tumour may be briefly described. The so-called *myxomata* are merely mucoid forms of glioma. *Melanotic* tumours have been met with, always associated with similar growths elsewhere.

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[*] See 'Medico-Chirurgical Trans.,' vol. lix, 1876, p. 217.

Fibroid tumours are very rare, and have been met with chiefly in the cerebellum or cerebellar peduncle, small in size, usually single, but in one case multiple. *Bony tumours* are usually partly fibroid, "osteo-fibromata." They have been met with chiefly in the cerebral hemispheres,—once in the cerebellum, once in the central ganglia, and once were multiple. (It must be remembered that *calcification* may occur in any degenerating tumour, tubercle, glioma, or sarcoma.) *Psammomata* are small tumours which may consist of fibrous tissue mingled with particles of "brain sand," such as occurs in the pineal gland or choroid plexus—calcareous particles, which are sometimes aggregated into larger irregular masses. These growths usually spring from the dura mater at the base of the brain, or from the choroid plexus, and form small hemispherical tumours, reddish grey, smooth, and hard. *Cholesteatomata* are small hard bodies, which are usually found in recesses in the base of the skull, and are composed of epidermoid cells arranged in concentric layers. They are perfectly innocent in this situation, but they have also been met with of considerable size, as large as a hen's egg or even as a fist, growing into the cerebral substance, and even arising within the hemisphere, in the central ganglia, or the pons. They are said to contain cholesterine and stearine; hence their name. Among the rarest of intra-cranial tumours are *neuromata*, small growths containing nerve-elements. *Lipomata* have been observed on the surface of the corpus callosum and corpora quadrigemina, in the ventricles and on the dura mater;* and *vascular* or *erectile* tumours have been met with, once or twice, in the substance of the cerebral hemispheres. Fœtal tumours, *teratoma*, are among the most rare.†

Cysts in the brain are generally the relics of an acute destroying lesion, hæmorrhage or softening; the effused blood or degenerated nerve-elements having, in the course of time, become absorbed. Such cavities have usually an irregular shape, and no well-defined wall. Simple serous cysts, of more regular form and with a fibrous wall, are sometimes met with in the membranes, the cerebral hemispheres, or the cerebellum; their origin is uncertain. Small cysts are often found in the choroid plexuses, and now and then these attain a considerable size. More frequently cystic cavities are developed in connection with morbid growths, especially glioma and sarcoma. Such a growth may contain one or more cavities within it, or a cyst may be attached to one side of a growth, or may occupy almost the whole area of the tumour, its walls only consisting of the new growth. We have seen that a glioma often breaks down in part, so as to form a cavity, containing débris of its elements; in process of time the débris may be removed, and it is probable that the cyst thus formed may enlarge by the flow of liquid into it. Such cystic tumours are met with especially in the cerebellum, occasionally in the white substance of the hemispheres, very rarely in other parts of the brain. Dermoid cysts have been already mentioned.

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* And (as fat enclosed in a fibrous wall) between the two layers of the anterior part of the falx (Rolleston, 'Trans. Path. Soc.,' 1892).

† In a case recorded by Hugo Beck ('Zeitschrift für Heilkunde,' 1884) a walnut-sized tumour occupied the position of the pituitary body, and contained bony and cartilaginous tissues and teeth. The patient was a woman aged seventy-four, and the tumour had caused no symptoms. Dermoid cysts containing hair have also been found in the lateral ventricle.

Parasitic cysts may be either hydatid or cysticercous, the former usually single; the latter, which are less common, are often multiple. Hydatid tumours have been found outside the dura mater, but their usual position is the cerebral hemisphere, either in the white substance or within the ventricles. Cysticerci are most common in the membranes or the cortex, but have been found in the central ganglia, the crus, pons, cerebellum, medulla oblongata, or at the base. Occasionally as many as fifty or a hundred cysticerci have been found in various parts of the brain.

The *pineal gland* may be invaded by tumours springing from the corpora quadrigemina, and has occasionally been found the seat o independent growths, usually firm and hard, attaining the size of nut or a pigeon's egg. In one case the body was the seat of a cystoi enlargement which occupied the whole of the third ventricle.

Tumours of the base of the brain deserve special mention. Th usually spring from the dura mater, sometimes from the bone. Sa coma and carcinoma are most frequent; less common are psammon cholesteatoma, syphilitic gumma, enchondroma, fibroid growth, a hydatid cyst. The petrous part of the temporal bone, or the du mater over it, is the part from which growths most frequently sprin the neighbourhood of the sella turcica is another common seat, a sometimes they originate in the posterior fossa, adjacent to the foram magnum. They may spring from the bone in this part (Fig. 13 They often attain a considerable size, even that of a hen's egg, a compress the superjacent brain. From their position, the cran nerves are frequently damaged by basal tumours, which in anterior fossa often compress or invade the optic chiasma and ner to the eyeball, and, in the posterior fossa, the pons (Fig. 140), medu and the nerves which arise from them. The fifth nerve and Gasseri ganglion are affected with especial frequency by the growths wh originate from the petrous part of the temporal bone.

Intra-cranial growths entail certain pathological effects, to wh their symptoms are due. (1) By the process of growth th destroy directly the adjacent nerve-elements. This destruction partly the result of pressure, partly the effect of the growth of morbid tissue-elements. In the infiltrating tumours, these tiss elements grow between and enclose, and gradually destroy, the ner elements. In the non-infiltrating growths the nerve-elements per before the compressing tumour, and the zone of softening around th

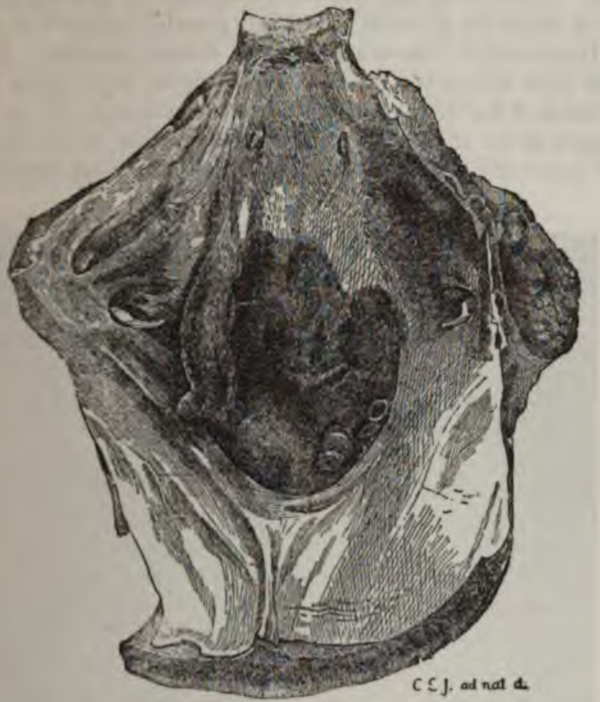

Fig. 139.—Growth from the occipital bone encroaching on the foramen magnum and compressing the medulla. (Drawn by the late Mr. C. E. Jecks.)

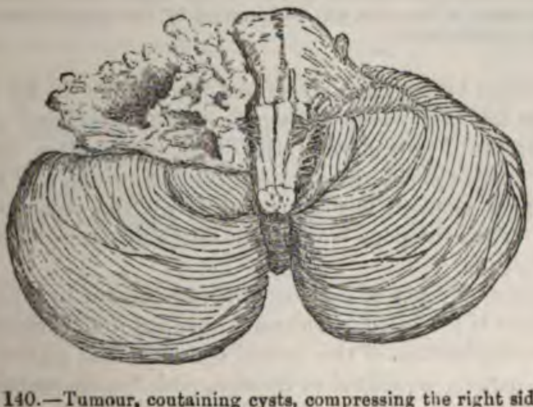

Fig. 140.—Tumour, containing cysts, compressing the right side of the pons Varolii.

growths is due to this destruction. In both cases, however, the slower the process of growth the less extensive and less complete is the damage. Nerve-fibres may persist within a slowly growing glioma, or may long resist the pressure of a slowly growing tubercle or sarcoma.

(2) Intra-cranial tumours also exert distant pressure. A growth is a new mass within the skull, which occupies more space than the tissue which it has destroyed, and so exerts pressure on all parts in that region of the skull. The nearer the parts are to the growth the greater is the effect of pressure upon them. The more the pressure is limited in range by resisting structures the greater is its immediate effect The falx offers some resistance to the extension of pressure from one cerebral hemisphere to the other, but is often displaced by it. More effective is the resistance of the tentorium, and tumours in the small space beneath it may compress all the structures therein contained. Thus the pons is often considerably damaged by tumours of the cerebellum.

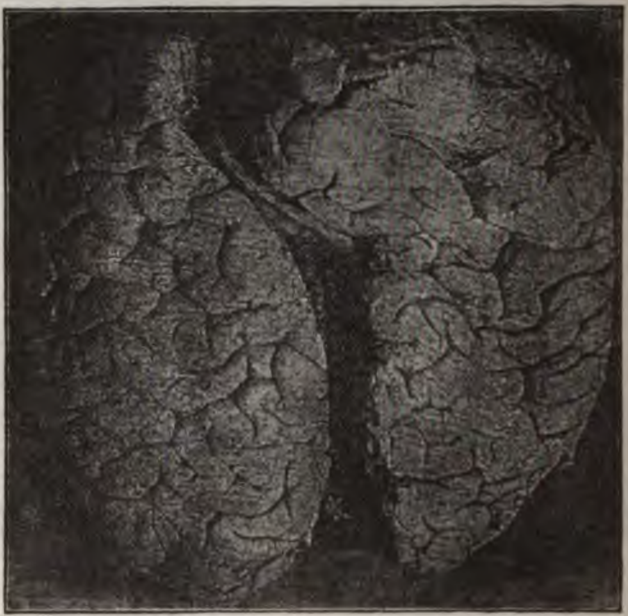

Fig. 141.—Large infiltrating tumour of the right frontal lobe causing displacement of the falx, and compression of the opposite hemisphere. (From a photograph.)

One pressure effect is of especial importance, that by which internal hydrocephalus is produced. The cerebro-spinal fluid is chiefly secreted by the choroid plexuses of the lateral ventricle, and passes thence by the third ventricle, aqueduct of Sylvius, and fourth ventricle. This passage may be closed by tumours of the pons, corpora quadrigemina, third ventricle, or of the middle lobe of the cerebellum, and the foramina of exit may be closed by secondary meningitis. The fluid is still secreted, but, unable to escape, distends the ventricles above the obstruction. Moreover, the veins of Galen, which return the blood from the choroid plexuses to the straight sinus in the tentorium, are often compressed by tumours of the middle lobe of the cerebellum or corpora quadrigemina. It is not probable that the compression of these veins will alone cause internal hydrocephalus, but it certainly increases the distension of the ventricles when the exit is closed. The convolutions are flattened, the bones become thin, and even the sutures of the skull may be separated by the powerful distending force.

(3) The growth of a tumour causes irritation, due in part to the pressure, in part to the vascular disturbance which attends it, in part, perhaps, to the influence of the process of new cell formation. The acute destruction of the nerve-elements is an irritative process, sometimes even inflammatory in nature. To this irritation the circumjacent softening is chiefly due, and by it some of the most characteristic symptoms are produced. But the irritation is not confined to the brain-substance. The meninges, especially the pia mater, are more prone to inflammation than any other intra-cranial structure. Hence, if the growth reaches the surface of the brain, there is usually distinct evidence of meningitis over it. The pia mater, if not invaded, is vascular, opaque, and thickened, and adhesions form between it and the dura mater. Traces of inflammation, recent or old, sometimes extend for a considerable distance from the tumour, often more in one direction than in another. Now and then there is evidence of meningitis at a distance, without any traceable connection with the growth, so that it must apparently be ascribed to distant pressure Thus in a case of tumour of the corpora quadrigemina, which had caused internal hydrocephalus, there were signs of inflammation beneath each orbital lobule, and nowhere else. In the diathetic forms of tumour, syphilitic and tubercular, meningitis is especially frequent and important, because it also results from the constitutional condition. In tubercular tumours the meningitis is usually general and acute, in syphilitic growths it is local and chronic. The tubercular meningitis is frequently the immediate cause of death in cases of tubercular tumour, especially in children. In syphilitic tumours, the adjacent pia mater and arachnoid are often extensively thickened by a mixed process of inflammation and growth, matting together the structures, and damaging nerve-roots at some distance from the growth.

An occasional effect produced by tumours of the brain is to cause a remarkable thinning of the cranial bones.* It is sometimes local, corresponding to the position of the growth; more often it involves the roof and sides of the skull, less commonly the bones of the base also. In extreme cases the bone may be reduced to the thinness of cardboard. Actual perforation has been observed only over the tympanum (Hale White). The inner surface of the bone is rough, in consequence of the unequal erosion. The outer surface of the dura mater may present corresponding inequalities, and the membrane is often thickened. The atrophy occurs when the tumour is within the substance of the brain as well as when it is at the surface, but the condition is only found when the tumour is large or there is internal hydrocephalus. It seems to be the result of the increase of intra-cranial pressure, which slowly causes an atrophy of bone similar to that produced by aneurism.

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* See, on this subject, a valuable paper by Hale White in the 'Guy's Hosp. Rep.,' vol. xliii, 1886.

SYMPTOMS.—The nerve disturbances produced by intra-cranial tumours are commonly grouped into two classes, *general* or *diffuse*, and *local* (see p. 74). The former are those symptoms which are produced by growths in various situations, and are of various kinds. Such are headache, vomiting, giddiness, general convulsions, optic neuritis, &c. The local symptoms (focal symptoms; German, *Herdsymptomen*) are those which depend on, and afford some indication of, the position of the tumour; they comprehend the local palsy and spasm, the affections of sensibility and of cranial nerves, which are the effects, more or less direct, of the existence of the tumour in a certain situation. It is important to remember that the division is not absolute. Some of the general symptoms may be produced to a greater extent by disease in one situation than in another, and so may have a focal significance (see p. 74).

In most cases the general symptoms precede the local, and the onset of both is usually slow and gradual. Sometimes they are trifling compared with the size of the growth, and in rare cases they may be absent, or, at any rate, so slight as to have attracted no attention, and a tumour is found post mortem, the existence of which was not suspected during life. This is especially the case with tumours springing from the membranes and merely compressing the brain, it may be to a considerable degree, but with slowness. Thus a globular fibroma, attached to the dura mater, although it was over the upper part of the central convolutions, two inches in diameter, and had caused a deep depression in the brain, produced no symptoms during life.*

Of the general symptoms, *headache* is the most constant, absent only in very rare cases. It is usually constant, with paroxysmal exacerbations; less frequently it intermits. In character the pain varies much; it may be dull or acute; sometimes it is described as rending, tearing, stabbing, boring. Its severity is usually such that sleep is more or less disturbed by it, and in the acute paroxysms the mind may be unhinged by the intense agony. It is usually increased by whatever causes passive congestion of the brain, as a cough, or muscular effort. It may be general, felt equally in all parts of the cranium, or chiefly in the front or in the back of the head, or on one side, or it may even be referred to a limited area. The locality of the pain does not always correspond to the locality of the disease. It may be referred to the forehead, when the disease is in the cerebellum, or to one side when the disease is on the other. But when the tumour is at the surface of the brain, the pain usually corresponds to the disease in situation. When the growth is in the white substance, the pain is often frontal, whether the disease is in the frontal or parietal lobe. When the disease is beneath the tentorium the pain is usually occipital, and often seems to pass down the neck. Unilateral occipital pain usually corresponds in side to the disease. If the tumour is superficial, gentle tapping causes pain over the seat of the disease, and not elsewhere. Neuralgic pain in the region of the fifth nerve indicates irritation of the nerve, and is not a "general" symptom; but this pain, when in the first division of the fifth, often blends with headache on the same side. The local pain produced by superficial tumours is doubtless due to irritation of the meninges. The more general headache has been referred to the influence of the increased intra-cranial pressure on the sensitive membranes, and the relief afforded in some cases by trephining, with or without puncture of the lateral ventricle, gives support to the opinion, but it does not explain the headache in all cases, since this may be severe when a small growth causes no increase of intra-cranial pressure, and the latter may be great without headache.

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* Willett, 'Trans. Path. Soc.,' 1892.

Optic neuritis occurs in a large proportion (probably about four fifths) of the cases of intra-cranial tumour, whatever be the seat or nature of the growth, whether it commences in the brain or membranes. Nevertheless it is certainly less frequent when the growth is in the membranes over the convexity, and merely compresses the brain, than when the cerebral tissue is actually invaded. The size of the tumour, *per se*, seems to have little influence, and the optic neuritis is certainly not produced simply by the mechanism of increased intra-cranial pressure. It has been caused by a tubercle of the pons no larger than a cherry, and was absent in a case which I watched throughout, in which a sarcoma, the size of the closed fist, growing from the dura mater, had compressed but not invaded the middle of one hemisphere, and must have raised the intra-cranial pressure as much as it is ever raised by the direct agency of an intra-cranial growth. It is probable that the neuritis is due to more than one mechanism, which vary in relative degree in different cases. One of these is the extension of a process of tissue irritation to the optic tract and nerves which, reaching the papilla, lights up a more considerable inflammation. Another is the distension of the sheath of the optic nerve and lymphatic spaces of the papilla, by subarachnoid fluid, containing, it may be, irritating products; possibly this determines the intensity of the inflammation. A third is the meningitis which, as we have seen, often occurs in cases of intra-cranial tumour, and may extend directly to the optic nerves. The neuritis is usually a transient event in the course of the tumour, which may exist for a considerable time, even for years, without any affection of the optic discs, and then neuritis may be rapidly developed, run its course, and pass into atrophy, and the symptoms of the tumour go on as before. Often, however, the occurrence of the neuritis coincides with an obvious increase in the other symptoms of the growth, and probably always indicates progress of the disease. In the majority of cases both eyes are affected, one sometimes more than the other. Rarely the neuritis is unilateral, and this although the disease may be one which usually causes bilateral neuritis; it may be on the side of the tumour or opposite to it.

The course of neuritis varies considerably in different cases. It may develop rapidly, with extravasations and great distension of veins, and subside in a few weeks to "consecutive atrophy." On the other hand, it may develop slowly, without distension of the veins, and be stationary for months, or even for a year, before it subsides. The course of the neuritis is some indication of the course of the tumour. Although a rapid neuritis is sometimes developed in the course of a slowly growing tumour, a chronic neuritis never results from a rapidly growing tumour. If the neuritis is acute and the tumour is not influenced by treatment, the neuritis usually goes on to atrophy. It often does so also in the chronic form, but now and then a slight neuritis, slowly developed, may subside, although the tumour continues to increase. But if the morbid process within the skull can be influenced by treatment, the neuritis may subside and leave little trace of its presence, and the recovery of normal vision may be perfect. The diminution in the intra-ocular affection may be the first indication of the improvement that is taking place within the skull. For other particulars regarding the mechanism, symptoms, and consequences of neuritis, the reader is referred to the special account of "Optic Neuritis," but it may be again pointed out that sight may be unimpaired as long as the inflammation is moderate in degree, and often fails much more while this is subsiding than during the active stage. The stage of atrophy is never reached until after many months, and this fact often affords very important indications. A tumour, for instance, may have caused marked symptoms for only a short time, but the condition of the optic disc may prove that the growth must have existed for many months before the other symptoms were manifest.

Mental Disturbance.—Stupor and coma are common as terminal phenomena in most forms of intra-cranial tumour, immediately preceding death. Apart from this final state, during the course of the disease, psychical symptoms are not unfrequent. The most common form is simple mental failure, loss of memory, depression, sometimes with emotional mobility, now and then varied by excitement. This condition may accompany tumours in any situation. It is sometimes, in tumours of the mesencephalon, produced through the agency of internal hydrocephalus compressing the cortex. Occasionally there is more pronounced mental disturbance, hallucinations and delusions, or actual dementia, in some cases simulating simple insanity. The tumour is then generally in the anterior portion of the frontal lobe, or in the temporo-sphenoidal lobe. A man with a large tumour beginning in the temporal lobe had vivid hallucinations that his wife, who had died two years before, was committing adultery in the ward before his eyes. In another case of glioma of the frontal lobe the only symptoms were headache, optic neuritis, attacks of petit mal, and mental disturbance with childish acts. The patient would strip the bedclothes off other patients who were in bed, turn pictures to the wall, &c. It is important to remark, moreover, that cerebral tumour, like every other form of brain disease, may evoke, in predisposed persons, the manifestations of hysteria. This is an important and common event, to which attention has been of late frequently directed. Many errors of diagnosis have occurred in such cases; the unmistakable symptoms of hysteria have had their frequent consequences in causing the physician to overlook the symptoms of organic disease.

Vomiting is a common effect of tumour in all parts of the brain, especially in the medulla oblongata, the middle lobe of the cerebellum, and the corpora quadrigemina; it is rather less frequently caused by tumours of the cerebellar hemispheres, the pons, the base, or the central ganglia. It is still less common (occurring in about a quarter of the cases) when the growth is in the cerebral hemispheres.

Giddiness, constant or paroxysmal, attends tumours in various situations. It is sometimes produced through the agency of the paralysis of an ocular muscle, or derangement of the auditory nerve (see chapter on "Vertigo"). Without any distinct peripheral mechanism, it is most severe and frequent in cases of disease of the pons, corpora quadrigemina, cerebellum, and middle cerebellar peduncle. In the latter case it is sometimes very intense, and is frequently associated with a tendency to a corresponding motion of the head or body. It is less frequent in tumours of the cerebral hemispheres than in those of the central ganglia, but unsteadiness, like that of cerebellar disease, has been observed in tumours of the pre-frontal lobes.*

Affections of Speech.—A peculiar slowness of speech is sometimes met with in association with tumours in almost every position, but with especial frequency in tumours of the cerebrum. A tendency to separate the syllables is conspicuous in some cases of tumour of the pons. With mental dulness from tumours in any situation, it is not infrequent for words to be clipped, or syllables run together in "confluent articulation." Marked difficulty of articulation occurs chiefly when the growth is situated in the pons or medulla, or in the posterior fossa of the base. It is also produced, now and then, by tumours of the cerebellum when these compress the pons or medulla. Aphasic difficulties of speech are chiefly due to tumours of the left cerebral hemisphere, cortex or white substance; as a permanent symptom only when the growth directly damages the speech-centres already described (p. 109), when the impairment may be of the motor or the sensory type, but it is often much slighter than would be expected from the size of the growth, and is sometimes paroxysmal.* It occurs also as a variable and intermitting symptom in disease of neighbouring parts, even of the upper part of the central region, chiefly in association with convulsions, and probably as an effect of irritative inhibition.† It may thus be caused indirectly by growths still lower in various parts of the central ganglia and even in the pons; the defect of speech mentioned on p. 318 was caused by a tumour.

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* Bruns, 'Neur. Cent.,' 1891. The cerebellum is connected with these.

Motor Disturbance.—Paralysis occurs—(1) Under the form of ordinary hemiplegia, almost always gradual in onset, affecting the lower part of the face, the arm, and the leg, from tumours situated in, or pressing on, the upper part of the pons, crus, internal capsule, or the cortex. Its degree and extent vary much, according to the slowness or rapidity with which the pressure is exerted.

(2) Partial hemiplegia, paralysis of the arm, or arm and face, less commonly of the leg, is usually the result of growths in or beneath or adjacent to the motor part of the cortex, and is often associated with convulsions beginning locally on the paralysed side. Both the local palsy and the local convulsion usually commence in the extremity of the affected limb, and have the same significance. The position of the growths causing such limited symptoms has been already described in the chapter on "Localisation."

When the tumour is seated below the hemispheres, in the crus, pons, or medulla, the hemiplegia is often accompanied by paralysis of one or more cranial nerves on the side corresponding to the tumour, and opposite to the affected limbs, as described at p. 317.

Basal tumours only cause hemiplegia when they are seated, or extend, so far back as to compress the crus, the pons, or medulla; and the character of the hemiplegia is similar to that produced by tumours in these parts, but the nerve-palsy is usually more extensive, and occurs earlier, than in the case of growths within the brain-substance.

(3) The paralysis produced by intra-cranial tumours is sometimes not unilateral, but bilateral. This may result (*a*) in rare instances from symmetrical tumours on both sides of the brain, generally in the central ganglia or white substance; one tumour may be larger than the other, and may cause the chief symptoms; (*b*) from a single tumour in such a position that it compresses the motor tracts of both

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* *E.g.* a cyst in the left temporo-sphenoidal lobe caused frequent long attacks of unconsciousness, followed by inability to "find the desired word." (Franks, 'Brit. Med. Journ,' 1888.)

† It was probably by this mechanism that a solitary tubercle in the right lower motor region of a right-handed patient caused aphasia. (Oppenheim, 'Neur. Cent.,' 1890.)

sides. This can only happen when the tumour is situated in or near the mesencephalon, where the two motor tracts are still contiguous, and it is generally due to a tumour in or compressing the pons or the medulla oblongata. In the latter situation the weakness may be chiefly in the legs, causing paraplegia, usually greater in one leg than in the other, but rarely limited to them, and distinguished by the affection of cranial nerves. When the damage to the motor tracts is by pressure from a distance (as in the case of tumours of the cerebellum or corpora quadrigemina) the bilateral weakness is often slight in degree, but an increased knee-jerk and a foot-clonus are seldom absent. (The exceptional loss in some cerebellar tumours has been mentioned at p. 321.)

Contracture, persistent tonic spasm, with excess of myotatic irritability, is frequently associated with the paralysis in the limbs, and has no special significance. Confined to one limb, it usually indicates that the disease is in or near the motor cortex. The chief difference from the contracture which follows other lesions is that it often accompanies, instead of following, the loss of power, doubtless because the damage to the motor tract is not only gradual, but irritative in its character, and descending degeneration occurs readily. From the same cause the contractured muscles often waste early, but in moderate degree, the more irritative degeneration of the pyramidal fibres involving the finer nutrition of the spinal motor nerve-cells (see p. 84). General tetanic rigidity, without paralysis, may occur from tumours of the pons or cerebellum; in the latter case there is occasionally retraction of the head, with rigidity of the neck, but it is uncertain whether this is caused directly, or through the agency of basal meningitis, or of pressure on the medulla oblongata. Bilateral tumours in the cerebral hemispheres, involving the paths for the muscles of the neck and trunk, have also caused rigidity of these parts.*

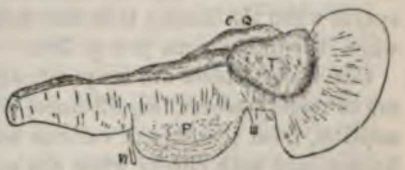

FIG. 142.—Longitudinal section of pons (P) and left crus cerebri, showing a tubercle (T) in the crus, beneath the corpora quadrigemina (C Q). The patient was a child, and the first symptom was jerky inco-ordination of the right arm exactly like that of disseminated sclerosis, followed by palsy of the arm, then of the leg and face, then of the left third nerve (III), and later of the right also.

Spontaneous choreoid or "athetoid" spasm also occurs in association with hemiplegic weakness and contracture, but less commonly than after some other cerebral lesions. It is accompanied by a slow spastic inco-ordination. It has been sometimes observed in cases of tumour of the parietal lobe, and of the neighbourhood of the optic thalamus. One form of inco-ordination in the arm is not at all infrequent in cases of tumour (especially tubercle) of the pons or crus, as in the case shown in Fig. 142. It is a coarse, jerky inco-ordination, precisely resembling that which is seen in disseminated sclerosis.* The limb, apart from voluntary movement, is free from spasm. This symptom, in disseminated sclerosis, seems to be due to the wasting of the white substance of the nerve-fibres, by which the conducting power of their axis-cylinders is lessened unequally. The pressure of a tumour on the motor tract may cause a similar interference with structure, and the phenomenon, which is certainly identical in aspect, may thus be due to a similar mechanism in each case. A finer tremor is not an uncommon accompaniment of the movement of weak limbs, and has no special significance.

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* See Ormerod, 'British Med. Journ.,' 1889.

Unsteadiness in the upright posture, most marked on walking, is a common symptom in tumour of the cerebellum, especially of the middle lobe. The body sways like that of a drunken person, and the patient may tend to fall forwards, backwards, or to one side. The movements of the arms are usually steady.

A tendency to the assumption of forced positions, or to certain involuntary movements, is occasionally seen. When there is vertigo the patient may tend to one side in walking, irrespective of the cause of the giddiness. A tendency to rotation of the body has been noted as an extremely rare symptom of tumour of the middle peduncle of the cerebellum. Rotation of the head to one side, with corresponding conjugate deviation of the eyes, occurs in some cases of cerebral tumour. Deviation towards the side of the lesion, so frequent at the onset of cerebral hæmorrhage, occurs in tumours of various positions, although not frequently. Occasionally there is a deviation from the side of the cerebral lesion, due to irritation and spasm when the disease is in the cerebral hemispheres, or to paralysis when it is in the pons (see p. 78).

Convulsive attacks are frequent, and are of four chief forms. Two are of merely general significance, occurring, like headache and optic neuritis, from tumours of various seat; while two are of local significance, met with chiefly when the growth occupies certain situations.

(1) Attacks of general convulsion with initial loss of consciousness, resembling the fits of ordinary epilepsy, occur in tumours of every part, but are not common when the medulla oblongata or cerebellum is the seat of the disease. They are more frequent in tumours of the posterior fossa of the base than in tumours of the anterior or middle fossa. The mechanism by which these convulsions are produced is uncertain. They are often ascribed to the general increase of intra-cranial pressure caused by the growth, on the ground that similar convulsions are produced in animals when the intra-cranial pressure is raised (to 130 mm. of mercury, Leyden). But it is scarcely justifiable to conclude that, because a rapid increase in the pressure causes convulsions, these are, therefore, thus produced by a slowly growing tumour; and the expla-

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* It is exceedingly probable that some cases published as examples of disseminated sclerosis in childhood have really been cases of stationary tubercle of the brain.

nation is not easy to reconcile with many clinical facts, which show, first, that large tumours do not necessarily cause convulsions; and, secondly, that even in the most severe form such convulsions may be produced when there is no increase of intra-cranial pressure. An instance of the latter was afforded by a case in which general convulsions persisted after other symptoms of syphilitic brain disease had been removed by treatment, and yet the patient actually died from the violence of the fits. The only disease discovered was a small, shrunken gumma in the anterior part of the right frontal lobe.

(2) Attacks of brief loss of consciousness, resembling the *petit mal* of epilepsy, occur occasionally, but not frequently, also from tumours in various situations, especially in the cerebral hemispheres. They are often associated with the general convulsions just described, and, like them, are of only general significance.

(3) The convulsions may commence in a manner which indicates that the discharge begins deliberately at some one part of the brain, and remains limited to it, or slowly spreads to other parts. The characters and significance of such convulsions have been already described. They are generally due to a morbid growth in or near the motor region of the cortex, and especially the place in which is represented the part in which the warning of the fit begins, whether the commencement is by motion or by a sensation. If the convulsion commences in the foot, the tumour is probably in the upper part of this region; if in the face, it is probably in the lower part; if in the arm, it may be in or near the middle region. Commencement in the hand has, however, been observed when the tumour is not contiguous to the central convolutions, which must then be influenced indirectly. The commencement of the fit sometimes varies in a way not easy to explain, except as a result of "irritation" in the various structures towards which the growth is extending. The discharge begins less often in structures that are much damaged (so as to cause paralysis) than in those adjacent, and merely irritated; the loss of power indicates the actual seat of the growth more surely than the initial convulsion. Thus in one case in which some fits began in the face, some in the arm, some in the foot, the last only was permanently paralysed, and the tumour occupied the highest part of the central convolutions.* Fits which begin locally often leave transient weakness in the part first convulsed, and this, if the attacks are frequent, may persist during the intervals, passing away when they cease. The frequency of these local fits is sometimes very great. In the case just mentioned a hundred or a hundred and fifty fits sometimes occurred daily, and in the course of eleven months the patient had 17,000 fits (carefully recorded). Occasionally local clonic spasm may continue for several hours or even days.

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* A similar case has been recorded by Osler ('Amer. Journ. of Med. Science,' 1885, p. 31). The fits began in the left hand but the palsy was in the leg, and the tumour was just beneath the highest part of the ascending frontal convolution.

The local commencement is sometimes by a sensation, tingling, "pins and needles," or pain, in the hand or in the foot, as mentioned at p. 92. As already stated, this sensory commencement has the same significance as the motor commencement, and the part in which the sensory discharge begins is that in which the chief irritation exists; the *secondary* spasm is not of localising significance.

A cerebral tumour may cause fits which commence by a special sense warning. It is probable that, in these cases, the tumour is situated in the cerebral cortex, in, near, or beneath that part in which the special sense-centre is situated. Some instances of this are mentioned on p. 92.

(4) Attacks of brief tetanic rigidity with retraction of the head have been observed in some cases of tumour of the cerebellum. Like the persistent rigidity, it is uncertain whether they are due to the irritation of the cerebellum or to pressure on the pons or medulla.

Sensory Symptoms.—(1) *Loss.*—Motor hemiplegia from tumour is often accompanied with unilateral diminution of sensibility of various distribution and character, according to the seat of the disease, as described in a preceding section (p. 94). Well-marked hemianæsthesia, extending up to the middle line, and usually with impairment of all the special senses on that side, occurs in cases of tumour damaging the posterior part of the internal capsule and subthalamic region, a not uncommon seat of syphilitic growths. Affections of sensation are rare and trifling in tumours of the anterior and middle fossæ, of the base, the corpora quadrigemina, and the cerebellum. Bilateral loss may occur, like bilateral motor palsy, from growths in the pons and medulla.

(2) *Sensory irritation*, pain, tingling, and other paræsthesiæ often accompany the anæsthesia produced by cerebral tumour, just as spasm often accompanies the motor palsy, in consequence of the irritating character of the lesion. Pain in the limbs is most frequent in tumour near the sensory tract or the central cortex, and is often felt in the limb which is chiefly paralysed or convulsed. The pain may be increased by movement, active or passive, and local convulsions may be sometimes induced by movements that cause pain. Other paræsthesiæ are especially frequent in tumours of the pons and medulla.

Cranial Nerves: Olfactory.—Loss of smell has been noted in rare cases of tumour in various parts of the brain, but with singular infrequency in any part, even in tumour of the anterior fossa of the base. Its occurrence in tumour elsewhere, even in the cerebellum, has suggested that its loss may depend on an olfactory neuritis, analogous to optic neuritis; but this is a mere hypothesis, at present unconfirmed.

Optic Nerve.—The frequency of optic neuritis renders impairment of sight a very common symptom of intra-cranial tumours, irrespective of their seat. Apart from this affection, which has been already considered, sight may be damaged by tumours situated in or near the visual path. The characters of the loss, according to the seat of the disease, have been already described. Symptoms of irritation of the optic nerve are extremely rare, except as the aura of convulsions, in tumour of the occipital lobe.

Third Nerve.—Isolated paralysis of the whole of one or both third nerves occurs only in tumours of or adjacent to the crus cerebri, first on the side opposite to the hemiplegia. Both third nerves may be affected by an interpeduncular growth, as probably in the case shown in Fig. 95, p. 181. Partial paralysis usually involves chiefly the levator or the internal rectus.

The *fourth nerve* rarely suffers alone, and chiefly in tumours in or compressing the corpora quadrigemina.

The *sixth nerve* is frequently paralysed alone, on one or both sides, by subtentorial disease, which exerts pressure on the nerve on account of its long and exposed course. It is also damaged, together with other nerves, by tumours of the pons, medulla oblongata, and in the posterior fossa of the base.

Combined paralysis of ocular muscles, slight and various, occurs in tumours of various situation, that exert pressure on the base —tumours of the deeper parts of the hemispheres, corpora quadrigemina, pons, and basal tumours. All the muscles of one eye are only paralysed by growths in the neighbourhood of the sella turcica, which usually cause also loss of sight.

Conjugate deviation of the eyes and head towards the side of a cerebral lesion (paralytic) or from it (spasmodic) has been already described. Its characteristic is that the axes of the eyeballs remain parallel in all positions. It must not be confounded with their associated paralysis, caused by tumours within the pons, described at p. 186, in which the external rectus on one side and the internal rectus on the other are affected together, so that the eyes cannot be moved towards the side of the lesion beyond the middle line. The variations in this symptom, and their localising significance, have been already considered. Tumour is by far its most common cause. Defective movement upwards or downwards is sometimes observed in cases of cerebral growths (as well as in those of the mid-brain), but its exact significance is uncertain (see p. 185). Nystagmus is an occasional symptom of tumour in many parts, but is not frequent in tumours of the cerebellum. An unsymmetrical position of the eyes, one directed upwards and inwards, the other outwards and downwards, has been observed as an extremely rare symptom of tumour of the middle peduncle of the cerebellum. The pupils are often unequal, dilated or contracted, but alone their irregularity is of little significance, except as evidence of some intra-cranial disease.

The *fifth nerve* is almost constantly affected in tumours of the middle fossa of the skull, frequently in tumours of the pons and of the posterior fossa, occasionally and in slight degree from growths in the cerebellar hemisphere which exert distant pressure. Both the sensory and motor parts of the nerve are usually affected (especially by tumours outside the pons); sometimes the sensory only, rarely the motor part alone. All divisions of the nerve may be involved, or only the upper and middle. Neuro-paralytic ophthalmia is frequent from basal tumours, which damage the Gasserian ganglion, but the eye often escapes when the tumour is situated within the pons. The gradual damage to the sensory part usually causes irritation; and neuralgic pain, sometimes most intense and accompanied by hyperæsthesia, precedes loss of sensibility. Taste is often also lost.

Facial Nerve.—Paralysis of all parts supplied by the facial nerve, with the reaction of degeneration in the muscles, occurs only in tumours of the pons or posterior fossa, very rarely, and in slight degree, from the distant pressure of a cerebellar tumour, and is associated with paralysis of the limbs on the opposite side. Sometimes both facial nerves are paralysed. The significance of association of paralysis of the facial and other nerves is described at p. 245.

Auditory Nerve: Hearing.—Deafness is an occasional symptom in tumours of the lower part of the pons, of the medulla oblongata, and of the posterior fossa of the base. Its localising significance has been mentioned at p. 260. Bilateral deafness has been produced by growths in the corpora quadrigemina which had damaged the upper layer of the tegmentum, in which probably the auditory path ascends to the hemisphere. Subjective sensations of hearing have been produced by tumours of the pons and base, and also by growths in the temporal lobe. In the former case they are referred to the ear of the same side, in the latter to the opposite side, and may occur in paroxysmal form as the warning of epileptiform fits. The sounds produced by cortical tumours are, as a rule, not "elaborate" in character.

The Hypoglossal Nerve.—Besides the general relations of palsy of the tongue mentioned at p. 302, the nerve may be paralysed on the side opposite to the hemiplegic limbs by tumours of the posterior fossa of the base, sometimes by tumours of the pons, and especially by those of the medulla oblongata. The paralysis is sometimes an early symptom of a morbid growth on the front of the occipital bone, or of one springing from adjacent structures and extending into the foramen magnum. In these cases there is often conspicuous wasting of the paralysed half of the tongue.

The Spinal Accessory nerve is also occasionally paralysed from tumours in or near the medulla, especially by those just mentioned as involving the hypoglossal nerves. Only those outside the medulla impair the action of the muscles of the neck, but more frequently the vocal cord on one side is paralysed; much less commonly both vocal cords are involved. The palate usually, and the tongue often, suffer at the same time. Bilateral palsies are occasionally met with, and the lips may also be involved, causing symptoms similar to those of progressive bulbar paralysis. Very rarely the same combination of symptoms has resulted from symmetrical tumours of the cortex in the lower part of the central convolutions.

The *pulse* is often infrequent in tumours anywhere within the skull, and the symptom has no special significance. *Respiratory* disturbance is chiefly met with in growths near the medulla, and these have also caused extreme frequency of the pulse.

The *Sphincters* are not often paralysed from intra-cranial tumour, and the loss of power over them is of no special significance. Untimely evacuation of bladder and of rectum is, however, very common in association with the mental apathy and dulness which are so common in tumours of all parts. An increased secretion of *urine*, and the presence in it of sugar and of albumen, have been met with in rare cases of tumour in various parts of the brain; glycosuria occurs most frequently in association with tumours of the pons and medulla, but not so frequently as to have any strong significance apart from other symptoms.

External Symptoms.—Intra-cranial tumours rarely manifest their existence except by the symptoms already described. Only those growths which spring from, or invade, the bone of the skull are recognisable externally; swellings on the surface of the skull occur in rapidly growing tumours springing from the dura mater. Growths in the bone of the middle fossa of the skull are sometimes recognisable on the under surface, in the palate. More commonly tumours of the anterior or middle fossa invade the orbit, and cause bulging forwards of the eyeball.

Regional Symptoms.—The symptoms of tumours in various parts of the brain are, in general, those that have been described in the chapter on "Localisation," p. 308, and need not be here repeated in detail. The chief deviation from the typical facts there enumerated arises from the comparatively slight degree of symptoms due to pressure, a deviation that is proportioned to the slowness with which the pressure is exerted. The effects of destructive invasion are always greater than those of compression.

The symptoms of tumours of the base, however, need brief enumeration. Growths in the *posterior fossa* cause nearly the same symptoms as tumours of the pons or medulla, subject to the same variations according to their seat. The most important difference is that the cranial nerves suffer earlier than the motor tract, and the combination of the nerves affected is somewhat different. Thus the facial and auditory suffer together from external pressure, rarely from internal disease. The sixth (abducens) is paralysed without the conjugate internal rectus, which suffers with it in disease of the centre. The combination of palsy of one half of the tongue, palate, and glottis is especially frequent from tumours beside the medulla. Neuro-paralytic ophthalmia, probably from implication of the Gasserian ganglion, sometimes occurs, but less frequently than in disease of the middle fossa. Lastly, pressure on the middle peduncle of the cerebellum often gives rise to intense vertigo, and an inclination to deviate to one side in walking. Convulsions occur in cases in which the pons is compressed, and are rather more frequent than when the tumour is within the pons.

Middle Fossa.—The course of the fifth nerve, and the position of the Gasserian ganglion, render it very liable to suffer in tumours of the middle fossa, and symptoms of its irritation and paralysis, including inflammation of the eyeball, often rapid and destructive, are frequent effects of disease in this region. From extension backwards of the growth, or by meningeal inflammation excited by it, the facial and auditory nerves may likewise suffer. Other nerves are rarely affected unless the growth is situated near the sphenoidal fissure, when all those to the eyeball may be involved. Hemiplegia is rare and slight, and is produced by the extension of the growth to the neighbourhood of the crus. Convulsions are extremely rare, and it is not common for there to be conspicuous mental symptoms.

Anterior Fossa.—Tumours are rarely limited to the anterior fossa of the skull, and the chief symptoms they produce are by their extension beyond it, backwards to the optic nerve (unilateral loss of sight), to the chiasma (temporal hemianopia, or irregular, even transverse hemianopia, probably from damage to all the higher fibres of the chiasma), and even as far as the crura, causing paralysis of the limbs, usually on one side only. From compression or invasion of the frontal lobes of the brain, mental symptoms occasionally result, and a peculiar childishness has been thought to have some diagnostic value. Loss of smell is common, in most cases limited to one side.

Tumours of the pituitary body cause usually vague symptoms, resembling those just mentioned, but frequently with failure of sight, and with indications of a lesion of the chiasma. Sometimes, however, the tumour causes very slight symptoms, and may run an almost latent course, even when the size attained is considerable.[*] Glycosuria has been sometimes observed.

Basal tumours may extend horizontally over a large area; occasionally a growth has occupied almost the whole of one half of the base of the skull. The symptoms, when the growth has been soft, have often been few, and a correct diagnosis has been impossible.

Multiple cerebral tumours sometimes cause only "general" cerebral symptoms, or one growth causes conspicuous disturbance, and there is no evidence during life of the existence of the others, or only equivocal indications of their presence. Occasionally two or more growths exist in the same part of the brain, and cause extensive but connected symptoms; this is especially the case with tumours in the posterior fossa of the base. On the other hand, tumours in different parts of the brain sometimes cause symptoms so distinct as to afford clear evidence of their separate influence.

COURSE.—The great characteristic of cerebral tumours is that their symptoms are gradual in onset and slowly progressive in course. The earliest symptoms vary, but are often general, and no one is so frequent as headache, although optic neuritis is sometimes a very early effect, especially of tumours of the cerebellum. Convulsions may first indicate the existence of brain disease, when a tumour is in or near the motor cortex; and they may be excited by some accessory influence in a manner that makes it easy to mistake the nature of the disease. The first fits due to a tumour of the right motor cortex followed a painful injury to the left arm, in which the fits began, with similar pain. Symptoms of irritation or paralysis of some cranial nerve often occur early, but hemiplegic weakness very rarely precedes headache.

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[*] *E.g.* case recorded by Cunningham, 'Journ. of Anat. and Phys.,' July, 1879.

Although the course of the symptoms is usually slow and gradual, it is rarely quite uniform, and exceptions to the gradual course are occasionally met with. In slowly growing tumours the progress may intermit, stationary intervals alternating with periods in which the symptoms increase. A rapid increase, followed by partial subsidence, probably results from secondary meningitis or local neuritis. The traces of past meningitis may be distinct after death, unless a long time has elapsed. In a case of tumour occupying the third ventricle, and compressing the corpora quadrigemina, the patient suffered, six months before death, from an attack attended with severe occipital headache, vomiting, and paralysis of the right fifth nerve, the left half of the face and tongue, the right half of the palate, and right vocal cord, and much difficulty in swallowing. These symptoms lasted for a few weeks, and then passed away almost entirely, slight weakness of the right masseter alone remaining. No evidence of meningitis could be discovered at the post-mortem examination six months later, but such transient symptoms of damage to nerve-roots extending as far back as the medulla oblongata could only have been due to intercurrent meningitis.

Hemiplegic weakness often follows unilateral convulsions as a transient effect of the "discharge," and is most marked in (and sometimes limited to) the limb in which the convulsion commenced. It may also follow, apparently as the result of inhibition, sensory discharges which do not cause spasm. But sudden and persistent hemiplegia may occur in the course of cerebral tumours without preceding convulsion. In rare cases it is the result of associated or independent vascular disease,* or of vascular occlusion from the effect of the growth on adjacent vessels. It may also be the result of a hæmorrhage into the growth, such as is common in the case of soft glioma. This is also rare, because the hæmorrhage is into, and not outside the tumour, and does not cause paralysis unless the growth occupies the motor region, but a growth so situated has usually caused paralysis from its size, before the hæmorrhage occurred. Lastly, sudden hemiplegia occasionally occurs in the course of a cerebral tumour, without there being any mechanism discoverable after death by which the sudden onset can be explained. The fact is important, its explanation obscure.*

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* As in the case of a man who had a firm tumour three quarters of an inch in diameter below the floor of the left lateral ventricle, and had independent hæmorrhages in both right and left corpora striata (Mitchell, 'Edin. Med. Journal,' Nov., 1883).

In the majority of cases, the progressive course of an intra-cranial tumour ends in death. But the rapidity of progress varies much, according to the character and position of the tumour; the former determining the rate of growth, the latter the symptoms which the increase in size produces. Thus a tumour of the same kind and size in the anterior part of the cerebral hemisphere may cause far slighter symptoms than if seated in the pons. Other things being equal, the slower the growth of a tumour, the slighter are the symptoms, since the slower the pressure, the better is it borne.

The duration of the symptoms varies from a few weeks to many years, but it is generally between six months and two years. The most rapid cases are tubercle, soft sarcoma, cancer, rapidly growing glioma, and syphiloma. The cases of longer duration are tubercle, firm sarcoma, slowly growing glioma, and the various hard tumours, fibroma, enchondroma, &c. Tubercles furnish some of the cases of shortest and also of longest duration.

The most common causes of death are—(1) Exhaustion, induced especially by the violence of the pain, and the interference with nutrition entailed by the frequent vomiting, and the difficulty of feeding occasioned by mental dulness or by dysphagia. (2) Coma coming on gradually, and usually due to the general increase of intra-cranial pressure, sometimes also produced by hydrocephalus. The lower centres participate in the apathy of the higher, and from the lowered sensibility of the respiratory centre, mucus accumulates in the lungs, and the resulting interference with respiration is generally the immediate cause of death. (3) Death is sometimes sudden. This occurs most frequently in tumours of the medulla oblongata, almost as frequently in tumours of the cerebellum, next in tumours of the central ganglia, occasionally in tumours of the cerebral lobes, corpora quadrigemina, pons, and base, but rarely in tumours of the cortex (Bernhardt). It is apparently sometimes due to pressure on the respiratory or cardiac centres of the medulla, sometimes to the mere intensity of exhausting symptoms, while it occasionally occurs in a mysterious way, without discoverable mechanism. (4) Lastly, death may occur from some other effect of the cause of the tumour. There may be intra-cranial or tubercular meningitis in tubercular tumours of the brain, or syphilitic disease of the vessels in cerebral syphiloma. Other fatal effects of the cause of the tumour are extra-cranial, as general tuberculosis in cases of tubercular tumour, or cancer elsewhere when there is a secondary cerebral growth. The subjects of very chronic tumours often die from intercurrent unrelated diseases.

But cerebral tumours are not invariably fatal. Syphilitic growths are more amenable to treatment than any other form of intra-cranial disease due to the same cause; the symptoms often pass away, and usually lessen, the degree of improvement depending on the extent to which they are due to interference with function by pressure, and not to actual destruction of nerve-elements. Symptoms of irritation, such as convulsions, may, however, persist in spite of treatment, the nerve-elements retaining the functional disposition resulting from the irritative damage, although the cause of this has been removed. Tubercular tumours may also cease to grow, and, as already stated, may become inert, and the symptoms may not only cease to increase, but even lessen, from the recovery of the nerve-elements damaged only by irritation or by pressure. When arrest is once obtained, the symptoms may remain stationary for an indefinite period. It is certain that a similar arrest occurs occasionally in some other tumours, in which degenerative changes, often calcareous, take place, and the residual mass gives rise to no symptoms except those which result from the damage caused during its active growth. But the result is rarer than in tubercular growths. A girl of fifteen suffered from left hemiplegia, defect of sensation, left hemianopia, headache, and double optic neuritis, all of gradual onset, in the course of three months. Under treatment, first the neuritis subsided, then the headache, then the leg improved, and lastly the arm, but with the development of a spastic state in the latter, which has continued with the hemianopia; otherwise she has been now for six years perfectly well.* In this case it is probable that a tubercular tumour existed in or near the optic thalamus. In another case,† a girl of twenty-three suffered from severe headache, double optic neuritis, slight bilateral weakness in the limbs, and paralysis of the upward movement of both eyes, cycloplegia, vomiting, and some opisthotonic convulsions. All the symptoms disappeared except the headache, and for three years there was no recurrence; she then died suddenly. A tumour in the corpora quadrigemina or anterior part of the middle lobe of the cerebellum had been assumed to exist, and a small degenerated growth was found in the front of the valve of Vieussens. I have seen a considerable number of other cases with well marked symptoms of a tumour, probably tubercular, which passed away permanently under treatment.

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* I have elsewhere recorded some cases illustrating the phenomenon, "On Sudden Paralysis in Cerebral Tumour," 'Brain,' vol. i, 1879, p. 48.

Diagnosis.—The problem of the diagnosis of a case of cerebral tumour includes four questions: (1) Is there organic disease? (2) Is it a tumour? If so, what are (3) its seat, and (4) its nature? Of these questions, to the first an answer can be given almost always, to the second generally, to the third often, and to the fourth sometimes.

1. As evidence of organic disease the focal symptoms are of more importance than the "general" (diffuse) symptoms. Of the latter,

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* 'Medical Ophthalmoscopy,' Case 2, p. 282 (2nd ed.).

† Loc. cit., Case 52, 2nd ed.

optic neuritis is, alone, the most significant (although not in itself conclusive); next, vomiting without gastric cause; while headache, giddiness, and general convulsions are of significance chiefly when combined with other symptoms. Convulsions beginning locally are of the nature of "focal" symptoms, but are not alone conclusive of the existence of organic disease, since they may occur in idiopathic epilepsy. Both headache and giddiness are extremely common without organic disease; nevertheless headache is a suspicious symptom if so severe as to keep the patient awake at night, and severe vertigo is also suspicious if there is no indication that it depends on disturbance of the labyrinth. As a rule, other symptoms exist to give significance to those which, alone, are equivocal. Two conditions are most likely to give rise to diagnostic error. One of these is the presence of diffuse symptoms, due to a blood-state, which may simulate those produced by intra cranial tumour. Headache and optic neuritis may be produced together by three general conditions—anæmia, kidney disease, and lead-poisoning. In the first of these the poverty of blood is always extreme; other symptoms of anæmia are obtrusive; the patients are usually young girls; optic neuritis develops with a rapidity unusual in tumour; all focal symptoms are absent; and the headache and neuritis rapidly subside under the influence of iron, especially if the patient is kept at rest in bed, and is well fed with easily digested food. In kidney disease, in which there is conspicuous neuritis, this is rarely very great in degree, and a sufficiently minute scrutiny of the retina will usually reveal degenerative changes, only caused by neuritis alone if it has been intense.* The urine contains not only albumen (which may be present also in cerebral tumour), but also casts; while the tension of the pulse, and hypertrophy of the heart, add significance to the other symptoms. Attention to these points, and to the absence of signs of focal disease, will usually prevent an error, in spite of the fact that headache is often considerable in these cases. The cerebral symptoms which occur in lead-poisoning—headache, optic neuritis, delirium, and convulsions—would be very liable to mislead if it were not that they are almost invariably preceded by other symptoms of saturnism, too conspicuous to be overlooked, and too distinctive to mislead.

The second condition, likely to give rise to error, is that in which a partial functional affection of the brain causes symptoms which simulate those of organic disease. Hysterical hemiplegia, spasm, contracture, or anæsthesia may be thought to be due to such a lesion; the more readily if headache or vomiting accompany them. The principles of their distinction are considered in the chapter on hysteria. The unilateral affections possess characters of their own, which differ from those of organic disease, and they often come on after emotional disturbance or a hysteroid convulsion, or in those who have watched the genuine symptoms of organic disease. The absence of a one-sided alteration of the reflexes, and of optic neuritis, constitute additional evidence, although of less weight, because of negative character.

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* Compare 'Med. Oph.,' pl. viii, fig. 2.

It is much more frequent for the symptoms of a tumour to be ascribed to hysteria, either because the patient is of the female sex, or because definite hysterical disturbance is evoked by the organic disease. A mistake is, however, readily avoided by attention to the simple rule, which, mentioned already more than once, cannot be too often repeated, that symptoms of hysteria do not lessen the significance of any symptoms of organic disease, the absence of which should be definitely ascertained.

When convulsions are the chief symptom, the disease may be confounded with idiopathic epilepsy. The mistake is most likely to occur in cases of slowly growing tumour in or near the central convolutions; in one recorded case the fits recurred during fourteen years, and there were long periods of freedom. The fits which thus recur almost always begin locally, as "cortical epilepsy;" but local commencement, whilst it suggests organic disease, does not prove it. The most important indication is the presence of other symptoms besides the convulsion. Occasionally, in the absence of other symptoms, causal indications may be allowed weight; *e. g.* a history of inherited tendency to epilepsy on the one hand, or, on the other, of a cause of tumour, such as syphilis. The probability of epilepsy is increased if convulsions, existing alone, begin sometimes on one side, sometimes on the other, and also by the length of the time during which they have been the only symptom.

As stated above, the existence of organic disease can *almost* always be recognised. In rare cases, the presence of a tumour has been unsuspected until revealed by a post-mortem examination. This has been especially the case with tubercular growths; sometimes because the symptoms have been masked by those of some other grave disease, or are of a subjective character, like hemianopia, readily overlooked.

2. If the symptoms are such as to indicate the presence of organic disease, the second question presents itself,—is their cause a tumour? To this question their gradual mode of onset usually supplies a ready answer. In the rare cases of tumour in which sudden symptoms occur, these are always preceded by others of gradual development. The fact that the symptoms never attain a high degree in the course of a few hours excludes at once the common vascular lesions, cerebral hæmorrhage and acute softening of the brain. The other lesions of slow development are few and rare.

Cases of general paralysis of the insane can only be confounded with the rare cases of cerebral tumour in which symptoms such as mental failure, general weakness, and slow speech exist alone. The absence of headache, optic neuritis, vomiting, and the presence of the tremor in the muscles of the lips and face, and of exalted ideas, will generally suffice for the diagnosis.

The very rare forms of chronic inflammation of the brain (see p. 468) may cause symptoms identical with those that result from cerebral tumour; the distinctions, as far as they exist, have been already mentioned.

Intra-cranial aneurism is, in fact, a tumour, and often produces symptoms closely resembling those of a growth. The distinction is considered in the chapter on this disease.

Abscess of the brain sometimes causes symptoms which closely resemble those of tumour, and give rise to real difficulty. Headache, vomiting, mental dulness, and optic neuritis are common to the two diseases, but the last is rather less common in abscess than in tumour. Focal symptoms are also comparatively rare. Slowly progressing symptoms, of uniform course, are characteristic of tumour, while in abscess there is usually a rapid development of acute and grave cerebral disturbance, after a "latent" period, in which symptoms are trifling or absent. The presence of a cause of abscess (previous injury, ear disease, or suppuration elsewhere) increases the probability of its existence, but it must be remembered that injury may cause either a tumour or an abscess. The points of distinction between the two are described more fully in the chapter on the latter disease.

Acute meningitis can rarely be confounded with cerebral tumour. The only difficulty arises in cases of tubercular meningitis, in which tubercular growths in the brain co-exist, but cause few symptoms before the onset of the meningitis. One important indication in such cases is the degree of optic neuritis. That which results from the meningitis alone is rarely considerable, and if the swelling of the papilla is great, and hæmorrhages exist—especially if such a change is found soon after the onset of the symptoms of meningitis,—it is probable that there are tubercular growths; the more so if headache or vomiting had long preceded the onset of the symptoms of inflammation.

Occasionally an acute increase (or even apparent onset) of the symptoms may be occasioned by exposure to some general or special morbid influence—cold, the heat of the sun, or a blow on the head,—an influence which may conceivably excite meningitis, but the nature of the case may often be decided by the indications just mentioned. It must be remembered that tubercular tumours may run an almost latent course for a time, and may then cause symptoms so rapidly as to render the diagnosis a matter of great difficulty. In some cases a comparison of the relative degree and extent of the symptoms may help the decision. For instance, an ill-nourished child, five months old, came under treatment for right-sided weakness and rigidity, with complete paralysis of the left third nerve. These symptoms were said to have come on a week before, and the child was thought to be suffering from tubercular meningitis. But it was unlikely that so extensive a process as meningitis would completely paralyse one third nerve and leave the other cranial nerves unaffected. The symptoms pointed rather to a focal lesion of the left crus cerebri, probably a tubercular tumour, running at first a latent course; the diagnosis was verified a few days later. It must be remembered also that a tubercular tumour and meningitis often co-exist. In such cases time alone can render the diagnosis approximately sure. If symptoms continue for four or six weeks they are almost certainly due to a tumour.

Chronic meningitis is exceedingly rare except as a result of syphilis or alcoholism. In the latter case the symptoms are diffuse, and too slight to be really confusing, while the cause is commonly obtrusive. Syphilitic meningitis, when local, may be indistinguishable from tumour, except in the rate of the development of its symptoms.

Only those cases of cerebral tumour in which there is jerky inco-ordination can be confounded with disseminated cerebro-spinal sclerosis. A sufficient distinction is afforded by the fact that in sclerosis the inco-ordination is usually bilateral; in tumour it is always unilateral; in the former there is neither much headache, vomiting, nor considerable optic neuritis.

3. If the disease is a tumour, the next question is, where is it? The answer depends on the symptoms present, the localising significance of which has been already described, and need not be here recapitulated. It is important to remember that the diagnosis must depend, as a rule, on the grouping of symptoms rather than on the presence or absence of any one symptom. There is hardly any symptom which may not be absent, wherever a tumour is seated, and almost every individual symptom may be produced by disease in more than one position. It must also be remembered that, although the general region in which the growth is placed may be determined in the majority of instances, it is not often that its exact situation can be confidently affirmed.

4. The exact nature of the tumour can sometimes be determined with a high degree of probability, now and then with practical certainty. Much more frequently hardly more than a guess can be made—a guess that is almost as likely to be wrong as right. The most important indications are the following:

(1) The presence of morbid growths elsewhere, the nature of which can be determined. When symptoms of cerebral tumour are consecutive to an infecting growth elsewhere, *e. g.* cancer of the mamma, &c., it is practically certain that the intra-cranial disease is of the same nature. In rare cases the presence of an hydatid tumour in another part indicates that the cerebral tumour is of the same nature.

(2) The presence of a distinct general disease of which intra-cranial tumour is a common consequence. These diseases are tubercle and syphilis. In adults, the signs of phthisis are rarely absent when there is tubercular tumour of the brain, and their presence is strongly in favour of—their absence against—the tubercular nature of the cerebral growth. In children, however, signs of tubercular disease elsewhere

are often wanting, and their absence is of much less significance than their presence. The same is true of a family history of phthisis or tubercular disease. A physical configuration, such as often co-exists with a tubercular tendency, is also in favour of this nature of the growth. The symptoms or history of syphilis, congenital or acquired, render it highly probable that the tumour is of syphilitic nature. The absence of a history of constitutional syphilis, if the patient has had a chancre, should not receive much weight, because it is not uncommon for the secondary symptoms to have been absent or overlooked. Moreover, in an adult, the possibility of the syphilitic nature of the disease cannot be excluded, unless we can feel sure that the patient has never been exposed to the risk of infection in the ordinary way, and, in the case of a woman, the assurance must include her husband. Undoubted syphilitic disease is occasionally met with where there is no history or indication of primary or secondary disease, the former having been unnoticed, the latter absent or disregarded. But in both cases, of tubercular and syphilitic indications, the diagnosis is a matter of probability only, since a cerebral growth of different nature sometimes co-exists with either diathesis.

(3) The sex of the patient affords little indication of the probable nature of the tumour, and that afforded by age is very limited. If the patient is under fifteen, and presents no indication of inherited syphilis, the tumour is certainly not of syphilitic nature. If the patient is an adult, and presents no signs of phthisis, it is very unlikely to be tubercular.

(4) The help afforded by the seat of the tumour is also small, and is practically limited to the following facts. *Cæteris paribus*, if the disease is within the cerebellum or pons there is some probability that it is tubercle or glioma; it is unlikely to be syphilitic if in the cerebellum, but it may be if in the pons. If in the cortex, the probability that it is syphilitic is considerable, but is less if it is situated in the deeper parts of the hemisphere. A tumour outside the brain tissue is probably sarcoma.

(5) The course of the tumour is sometimes suggestive. A very slowly growing tumour in the hemispheres is probably glioma, and if the development of the symptoms is extremely gradual, it is most unlikely that the growth is syphilitic. A tumour which grows rapidly at the onset and then becomes stationary is probably tubercular. The occurrence of a sudden apoplectic seizure of moderate severity in the course of the symptoms is rare except in glioma.

(6) The effect of treatment is of diagnostic value only in the case of syphilitic and tubercular tumours. A disappearance of symptoms (especially headache, optic neuritis, and recent paralytic symptoms), under iodide of potassium and mercury, makes it highly probable that the tumour is syphilitic. Improvement under general tonics is some evidence that the tumour is tubercular, but the value of this indication is at present somewhat dubious, since the nature of most

tumours which thus improve is uncertain, and it is possible that other than tubercular growths may be thus influenced.

(7) Multiplicity affords little indication of nature, because tubercular, syphilitic, gliomatous, and cancerous tumours are multiple often or occasionally.

Prognosis.—Only when the tumour is of syphilitic nature can a reasonably good prognosis be given. Even in such a case it is only the disappearance of the diffuse symptoms, and of recent paralyses that can be confidently predicted. Palsy of more than a few months' duration may persist, although the growth is removed; and convulsions sometimes continue, but it is not uncommon for all symptoms to pass away. In tubercular tumours arrest is not uncommon, and the patient may live on indefinitely, for general tuberculosis exists less frequently than might be expected. The symptoms lessen, but in what degree depends on the seat of the disease; hemiplegia usually persists, but the symptoms of a cerebellar tubercle may disappear. In all cases a diminution in the headache and subsidence of optic neuritis which is moderate in degree (and has not, as it were, exhausted itself by its violence) usually precede other indications of improvement; they are therefore distinctly favorable signs, the recognition of which is very important. Conversely, the persistence of optic neuritis is of grave significance. These rules are, however (as we shall see), true only when no operation is performed.

In other forms of tumour the prognosis is serious. As a rule they progress to a fatal termination, and the probable duration of life can only be estimated from the rate of the progress in the past and present. It is likely, however, to be longer in tumours of the cerebrum or cerebellum than in those of, or pressing on, the pons or medulla. Considerable mental dulness, obstinate vomiting, severe and frequent general convulsions, apoplectic seizures, and rapidly developed and intense optic neuritis, are all indications which render the prognosis grave as to the near future. On the other hand, the absence of these symptoms indicates, other things being equal, that the danger is more remote. Very chronic optic neuritis is of especial significance as an indication that the course of the disease will be slow and prolonged. It is probable that, in most forms of tumour, arrest of growth now and then occurs, but it cannot be anticipated. Even when the growth of the tumour ceases, ventricular effusion may cause persistent general symptoms, which must be expected to continue unless relief can be afforded by the surgeon. The recent development of cerebral surgery has, however, improved the prospect in many cases, as regards the growth itself, to an extent that will appear from the next section.

Treatment.—The treatment of new growths, in such a position that they are beyond the reach of the knife, and of such a nature that they cannot be influenced by drugs, is always a sufficiently gloomy subject, and not least so when they are seated in an organ like the brain, in which they cause peculiar and varied suffering, and in which their development, even to a moderate degree, is seldom compatible with life. Too often all that can be done is to afford some slight relief to symptoms, and even this is frequently trifling in degree. The only case in which a considerable effect on the growth can be produced by drugs is that of syphilitic tumour. Whenever there is even a possibility that the tumour is syphilitic, iodide of potassium should be given in increasing doses up to half a drachm three times a day, followed, if the effect is inadequate, by mercury.

If it is probable or possible that the tumour is tubercular, general tonics are of paramount importance, and of these cod-liver oil, iron, and an adequate supply of food, are more often distinctly beneficial than any other agents. Cod-liver oil is of especial importance. Fresh country air is very useful, but does not, alone, exert such an influence as do tonics. A tonic treatment is indeed desirable in most cases; arrest of symptoms occasionally occurs under its influence when there is no reason to think that the tumour is tubercular. There is no evidence that any other drugs are capable of arresting the progress of morbid growths, and the chief practical question is the possibility of excision.

Apart from this problem is that of the relief of symptoms which we are powerless to remove. Those that depend on meningitis—which extend beyond the original range of mischief, and are characterised by subacute onset, with increased headache and vomiting, and often by fever—may sometimes be relieved by counter-irritation and by leeches, if the patient is in a condition to bear them. Of other symptoms, headache is one which most often calls urgently for mitigation. Relief can usually be obtained by the hypodermic injection of morphia, but the ready habituation to the narcotic renders its use admissible only in the most acute paroxysms. Indian hemp is sometimes useful, but hyoscine and especially antipyrin are more often effective. Sometimes counter-irritation to the neck gives relief to occipital pain, but no local application is so effective as ice, which often influences not only the pain, but the vomiting and convulsions. For the latter, bromide of potassium is less useful than chloral. In all cases, as much brain-rest as possible should be secured; active physical exertion, constipation, heavy meals, and alcohol should be avoided, since all these tend to increase the cerebral disturbance.

There remains the important question of operative measures, a subject which has become so extensive and complex as to have passed almost out of the domain of medicine. Only the salient features can, therefore, be noted here. Although it constitutes a step in therapeutic progress of signal interest, second to none in importance and in the power of saving life, it has limits that are quickly reached and may be easily overlooked. It is to MacEwen, Godlee, and especially to Horsley that we owe the discovery of the comparative safety with which even extensive operations on the brain may be undertaken, provided strict antiseptic precautions are observed. Without these, the danger is great of rapidly fatal inflammation; and even with them the dura mater cannot be opened without some risk of this event, or of fatal shock to the system. In all cases, therefore, this danger has to be weighed against the prospect of success in the removal of a growth, or the urgency of symptoms that may be relieved for a time, even when the tumour is beyond reach. It is only a small proportion of intra-cranial growths in which extirpation is practicable. A careful study of the details of 299 cases, by Allen Starr, has led him to the conclusion that, while 18 per cent. were accessible, not more than 6 per cent. could have been successfully removed. Earlier diagnosis probably renders this estimate too small, but it is certain that the majority of growths are beyond reach. The cases suitable for an operation are those in which the tumour is in or just beneath the cortex of the brain, and in such a locality that its position can be inferred with confidence, especially at the motor region, and probably also in the occipital or upper temporal lobes. Those that spring from the membranes and compress the brain offer the least difficulty. On the other hand, growths in the central ganglia, crura, pons, or base cannot be successfully removed, and they constitute 34 per cent. of the whole number. The frequency with which there is more than one tumour is another source of failure. A growth in one hemisphere of the cerebellum would no doubt be accessible, but such tumours seldom cause distinctive symptoms until they are of large size; even then it is scarcely possible to ascertain their exact situation, and attempts to remove a cerebellar tumour have hitherto been fatal. The middle lobe of the cerebellum is so inaccessible and so close to important structures that it is not likely that the removal of a tumour could be survived.

Very striking is the effect of the operation in some cases in which the tumour has rendered the patient unconscious, and has brought him apparently near the end of life. Immediate improvement has been followed by steady recovery, and in no malady has life been more distinctly saved. But such cases are unfortunately few, and we have to put on the other side the fact that the removal of an infiltrating tumour, even when perfectly accessible, involves also that of nerve-elements which still retain functional power, and therefore the operation is followed by increased palsy, which may be, in some degree, of permanent character. This is a minor consideration when life is threatened, but, in many cerebral tumours, the danger to life is not certain or near; the question of an operation is one of peculiar difficulty in many cases of the kind, especially when the tumour is possibly syphilitic or probably tubercular in nature. Each case must be dealt with according to its apparent character and obvious course, and the introduction of a new and greater danger to life must be carefully avoided. The danger is not yet fairly indicated by published facts. It must also be remembered that convulsions do not always cease when their original cause is removed; although their recurrence, after an operation, is occasionally only for a time, it is more frequently persistent.

But operations that fail to achieve their primary object are not, therefore, devoid of result. The removal of part of the cranial wall has been found, by experience, to be followed by a remarkable diminution, and even arrest, of the pain of a growth that cannot be excised, an effect which has generally continued as long as the patient's life endured, which is seldom, in such cases, for many months. Hence this operation has been advocated by Horsley as the surest mode of affording relief, apart from the possibility of excision. Pain is not, however, the only symptom thus relieved. The operation is usually followed by a remarkable subsidence of the optic neuritis, if this is present, probably because fluid effused outside the brain is no longer forced into the lymphatic spaces of the papilla, and no longer conveys to it material capable of intensifying inflammation that may descend the nerve. Hence it is probable that sight may sometimes thus be saved, even though a certain amount of neuritis persists. Against these advantages, however, must be placed the possibility that life may be materially shortened, and the urgency with which relief is required must be carefully considered. But the danger to life is less than from another procedure which has also been advocated for the same object, that of puncture of the lateral ventricles, any tender spot being chosen if the seat of the growth is uncertain.* This has so often set up a subacute inflammation, fatal in one or two weeks, that its wisdom is doubtful, especially in view of the fact that it seems to afford little, if any, more relief than the simple trephining. But if a growth can be influenced by drugs, their employment is obviously preferable to any operation, and, although the chance of systemic infection by a tubercular tumour has been thought to justify the removal of such formations, the danger of this infection seems, in practice, to be extremely small.†

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* Weir, Horsley, Knapp, and others.

† The above sketch deals only with the more obvious conclusions, suggested to the physician, regarding a subject which has largely passed into the province of surgery. As such, the reader will find it discussed in greater detail, and with much material for reflection, in the following articles. In these, and in many others that have appeared during the last three years, will be found a discussion on fundamental questions of the cranio-cerebral topography, of which some account is given in the early part of this volume, but which abounds with points that are still undecided.

Horsley, 'Brit. Med. Journ.,' 1890. MacEwen, ibid., 1888. Knapp, 'Intra-cranial Growths,' Boston, 1891. Fraser, 'A Guide to Operations on the Brain' (photographs), London, 1891.

INTRA-CRANIAL ANEURISM.

Both the larger arteries of the brain and their branches within the cerebral substance are occasionally the seat of aneurismal dilatation. Within the brain the aneurisms are minute, rarely exceeding a pin's head in size, and hence are called "miliary aneurisms." They are important chiefly as causing hæmorrhage into the substance of the brain, in connection with which they have been described. Only the aneurisms of the larger cerebral arteries will be considered in this section. Such aneurisms are more common in the vessels of the brain than in those of similar size elsewhere, partly on account of the considerable blood-pressure to which these vessels are exposed, and partly because their walls are more frequently the seat of morbid changes.

CAUSES.—Males suffer more frequently than females, in the proportion of 3 to 2. Practically unknown under ten years of age.* aneurisms occur in each decade of life, from ten to sixty, with nearly equal frequency; after sixty they become rare, but are met with occasionally up to extreme old age. About an equal number occur before and after forty, and about one sixth in the second decade of life. Thus they are much more frequent during the first half of life than are aneurisms elsewhere. An explanation for this is found in their immediate causes.

There appears to be, in rare cases, a peculiar inherited tendency to the formation of aneurism. A distinguished physician died young from the rupture of an intra-cranial aneurism; ten years later his brother, a medical student aged twenty, showed me a distinct aneurism on an artery of his hand.

The immediate cause is similar to that of aneurisms elsewhere—a change in the structure of the wall, whereby the muscular and elastic elements are replaced by distensible fibroid tissue. In consequence of this, the wall yields permanently under the blood-pressure. A bulging results, which goes on increasing, as the walls, thinned by extension, lose still further their power of resistance. The mechanism by which this structural change is produced varies in different cases. It may be a primary degeneration, or the effect of inflammation, caused by injury or imperfect embolism, or the structure may be changed by syphilitic disease. The two latter influences cause aneurism much more frequently in this than in other situations.

(1) *Primary degeneration* is an occasional cause in the second half

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* Yet it is possible that disease of the arteries due to inherited syphilis may cause aneurism in early life, since it may cause cerebral hæmorrhage. An instance in a boy of eight is mentioned on p. 389. The occurrence of aneurism from embolism is also possible under ten, although I do not know of any recorded instance.

of life. It may be a simple fibroid change due, apparently, merely to the strain to which the vessels are exposed, and this is especially influential in arteries which come off directly from large trunks. Thus an aneurism was seated on an abnormal artery which passed between the internal carotids beneath the optic commissure (Weir Mitchell). Atheroma seldom leads to simple dilatation, although the process of breaking down of the degenerated wall sometimes causes a dissecting aneurism. Fatty degeneration does not involve a tendency to yield before the blood-pressure, as does simple fibroid change.

(2) *Injury*, a blow or fall on the head, is an occasional antecedent (in 6 per cent.*), and seems to act sometimes by direct damage to the wall causing traumatic arteritis, sometimes by a change in the wall of the artery, produced by inflammation which spreads to it from adjacent structures. The internal carotid suffers most from this cause.

(3) *Syphilitic disease* affects, as is well known, the arteries of the brain more frequently than those of other parts. Its characters have been described at p. 425. A new growth infiltrates the wall, destroying the muscular tissue, and leaving cicatricial damage. The wall is often at last thinner and more opaque than normal, the cavity of the artery increased, and the vessel unduly distensible. An aneurism has been frequently met with in cases of constitutional syphilis in young adults, in whom no other cause was discoverable. It is, indeed, surprising that this consequence is not more common, when we consider how distinctly the aspect of the wall is altered, even in cases in which the disease has been removed by treatment. But it seems to be limited to cases in which the morbid process has existed for a long time, or has undergone spontaneous cicatrisation. It is possible that an aneurism only forms when the elastic lamina has been destroyed; this persists long after the muscular tissue. The basilar artery is a frequent seat of aneurism from this cause.

(4) *Embolism* is probably the most frequent cause in the first half of life. There are few cases during this period, without indications of syphilis or injury, in which there is not evidence of past or present heart disease.† We know that the fragments of lymph which are washed from the valves in endocarditis have often an irritative character, bearing septic material or organisms; and these, at the place at which they are arrested, may excite inflammation similar to that in their source. If an artery is imperfectly closed by such a plug, its walls become inflamed, altered, and yet are still exposed to the pressure of the blood, before which they yield. The mechanism has been traced in the arteries of the limbs, but, like embolic occlusion, is far more frequent in the arteries of the brain. Hence also the middle cerebral artery, embolic occlusion of which is the most

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* Eight cases of 131 collected by Killian.

† The frequency of heart disease was noted by Lebert and other observers, but the significance of the association was first pointed out by Church.

frequent, is that on which aneurisms are most common, and may even be multiple.* Aneurisms due to this cause may occur, however, on any artery, even the basilar or vertebrals. Complete closure is probably effective if a pervious branch maintains the circulation in the vessel on the cardiac side of the plug.

GENERAL PATHOLOGY.—Aneurisms are twice as common in the system of the internal carotids as in that of the vertebrals. The middle cerebral branches are affected more often than any other, but only a little more frequently than the basilar, the two together making up more than half the number of cases. Next in frequency is the internal carotid itself, which is, however, diseased only half as frequently as the middle cerebral. In two thirds of the cases of aneurism, one of these three vessels is the seat of the disease. The other arteries are affected in the following order of frequency: anterior cerebral, posterior communicating, anterior communicating, vertebral, posterior cerebral, inferior cerebellar.† Aneurisms of the arteries of the central ganglia, within the substance of the brain, are very rare. A remarkable instance, in which an aneurism in the lenticular nucleus attained the size of a small chestnut before it caused death by rupture, has been recorded by Bastian.‡ Other cases are on record in which an aneurism, although not found, was probable, because fatal hæmorrhage occurred under the conditions in which aneurisms are produced.§

Aneurisms are rather more common on the left side than on the right in the proportion of four to three. In about a fifth of the cases there is more than one aneurism, generally in consequence of embolism. Bilateral symmetry is very rare, as in the two middle cerebrals or the two vertebrals.* In character the aneurism is almost always a true sacculated aneurism, very rarely dissecting. Occasionally there is a general dilatation of the vessel. The walls are usually thin, but often are lined by laminated clot, and sometimes supported by thickened and adherent membranes. In size they commonly vary from a pea to a nut, sometimes attaining a larger size. They have been met with as large as a hen's egg on the internal carotid, anterior cerebral, basilar, and posterior cerebral, and as large as a pigeon's egg on the middle cerebral and posterior communicating arteries. In shape they are usually round, sometimes oval. The surface is red and smooth, and the tumour often has the aspect of a small round mass of clot, the wall being so thin as to be invisible. When rupture has taken place the aneurism is in the midst of a coagulum, and great care may be needed to discover it. It is best to wash away the clot slowly by a gentle stream of water.

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* Recent medical literature abounds with illustrations of the association of intracranial aneurisms and endocarditis. One striking instance may be quoted. A boy twelve years old suffered from mitral disease after rheumatic fever, and after death there were found circumscribed deposits on the mitral valve, with numerous infarcts in the spleen and kidneys. At the origin of the left posterior cerebral artery from the basilar there was a small aneurism the size of a pea, and the right posterior cerebral was closed by an embolus, immediately behind which was another aneurism of similar size. The artery beyond was obliterated. Here we have a demonstration of the local association of the two processes. (Rauchfuss, 'St. Petersburg med. Wochenschrift,' Feb. 18th, 1878, and 'Virchow's Jahresb.,' 1878, vol. ii, p. 102.)

† The following is the numerical frequency in 154 cases, obtained by combining, in so far as they do not overlap, the statistics of Lebert ('Berlin. klin. Wochenschrift,' 1866), Durand ('Des Anévrysmes du Cerveau,' Paris, 1868), and Bartholow ('Amer. Journ. Med. Science,' Oct., 1872), and the addition of ten cases recorded by others:—Middle cerebral 44 cases, basilar 41, internal carotid 23, anterior cerebral 14, posterior communicating 8, anterior communicating 8, vertebral 7, posterior cerebral 6, inferior cerebellar 3.

‡ 'Trans. Clin. Soc.,' 1884, p. 18.

§ Another instance is the case of hæmorrhage in a boy of eight, with inherited syphilis and arterial disease, mentioned already. A very instructive case of hæmorrhage into one corpus striatum, bursting into the ventricles, in a boy of fifteen, with slight mitral disease, has been recorded by Bastian ('Trans. Clin. Soc.,' xvii, 1884, p. 21). In each of these cases an aneurism of a cerebral artery was highly probable.

An aneurism, except it be of very small size, necessarily obtains room by compressing adjacent structures. The cerebral substance may be thus damaged, the nerves at the base compressed, and even the bone eroded. It is by this means that the chief symptoms are produced.

Rupture of the aneurism occurs in rather more than half the cases, and in a still larger proportion in the young. Most frequently the blood escapes into the membranes at the base of the brain. The opening in the aneurism is usually small, and the escape of blood may be gradual and even intermittent. It may be forced from the base into the fourth ventricle by the openings through which liquid escapes out of the ventricle; this distension of the fourth ventricle is occasionally the immediate cause of death. In some cases, however, in which the aneurism is supported by thickened membranes on its outer side, it gives way where it is in contact with, and perhaps embedded in, the brain-substance, and the hæmorrhage occurs, not into the membranes, but into the brain. Thus the blood from an aneurism of the middle cerebral may escape into the hemisphere until it reaches the lateral ventricle, and I have known an aneurism of the commencement of the posterior cerebral to tear up and infiltrate the pons before it escaped externally. The slowness of the hæmorrhage probably favours its extension into the brain, the blood slowly disintegrating the tissue before it, along the line of least resistance. In one case a narrow fistulous tract of blood extended from an aneurism of the middle cerebral, on the convexity, to the lateral ventricle. An aneurism of the internal carotid has been known to burst into the cavernous sinus.

General Symptomatology.—A small aneurism, in any situation, may cause no symptoms, and its existence may be unsuspected until rupture occurs. Such latency is most common in aneurisms of the anterior communicating and the cerebellar arteries, and it is not infrequent in those of the middle cerebral and basilar. When symptoms occur, these are partly general, partly local. Headache is the most common, often pulsating in character; it is usually continuous, but sometimes paroxysmal. Its seat has little relation to the position of the aneurism, except that it is usually occipital when the basilar artery is diseased. Giddiness is also common, whatever be the seat of the aneurism. Mental dulness and irritability have been caused by aneurisms in various situations, but most frequently when the anterior cerebral was diseased, least frequently by aneurism of the internal carotid and middle cerebral. Convulsions are not frequent, except in aneurism of the middle cerebral. Large aneurisms in any situation may cause paralysis of limbs, but this is most frequently due to those of the middle cerebral and basilar. The cranial nerves are often paralysed, as they are adjacent to the vessels most frequently affected (see Fig. 143). Optic neuritis is not common; it has been occasionally met with, but only in aneurism of the internal carotid; when present it is double. Probably an ophthalmoscopic examination would have revealed it in a larger proportion of cases. A murmur, the most common symptom of aneurisms elsewhere, is very seldom produced by those within the skull. In a few recorded cases* of aneurism of the internal carotid, not only has the patient been conscious of a murmur, but this has been distinctly audible on auscultation of the skull, and has been arrested by pressure on the carotid. In one case of aneurism of the left vertebral artery, beside the medulla oblongata, a loud murmur could be heard on each side, between the mastoid process and the spinal column.†

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* Mott, 'Brain,' 1889; Hale White.

When embolism causes an aneurism the obstruction is necessarily partial, and, as a rule, the causal process produces no symptoms. Nevertheless it is possible that transient symptoms sometimes occur at the time of the embolism. In the case recorded by Bastian, mentioned in the note at p. 531, three years before the fatal apoplexy, the boy was suddenly convulsed, and afterwards screamed for two or three hours. A careful inquiry might elicit a history of transient symptoms in many of these cases.

Aneurisms of Special Arteries.—The symptoms produced by aneurisms of special arteries depend on the structures adjacent to these vessels. Their relations are imperfectly presented in the brain after it has been removed. Fig. 143, representing the chief relations, is from a dissection, made at my suggestion by Mr. Shattock, for the purpose of showing the relative positions of the chief arteries, nerves, and other structures at the base of the brain. A reference to it will render clearer many points in the special symptomatology of intra-cranial aneurisms.

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* Coe, Holmes of Chicago, Hutchinson, Humble, Jeaffreson.

† Moser, 'Deut. Arch. f. kl. Med.,' Bd. xxxv, p. 418.

Internal Carotid.—An aneurism may be formed within the cavernous sinus, or after the artery has emerged from this, as it lies on the inner side of the anterior clinoid process and origin of the optic nerve, with the root of the olfactory nerve in front of it. The aneurism may compress the adjacent frontal and temporo-sphenoidal lobes, but its chief symptoms are due to compression of the optic nerve on the inner side, and of the nerves in the wall of the cavernous sinus on the outer side. The sight of the eye on that side fails. The third nerve usually suffers early, and ptosis may be the first symptom; but all the muscles may ultimately become paralysed, and the eyeball may lose sensibility in consequence of damage to the ophthalmic branch of the fifth. Smell also may be impaired. When the aneurism is large, and extends backwards, it may cause hemiplegia by pressure on the crus. Frequently, the pressure-effect is limited to the ocular nerves Rupture has occurred in less than half the cases. Transient fulness of the retinal veins has been observed to result from the compression of the cavernous sinus by a carotid aneurism, but the pressure is quickly relieved by the free communication of the ophthalmic and facial veins; the enlarged angular vein may be conspicuous beneath the skin. Optic neuritis, when it exists, is apparently due, not to the pressure on the sinus or the nerve, but to the extension of inflammation in the vicinity of the aneurism (Michel).

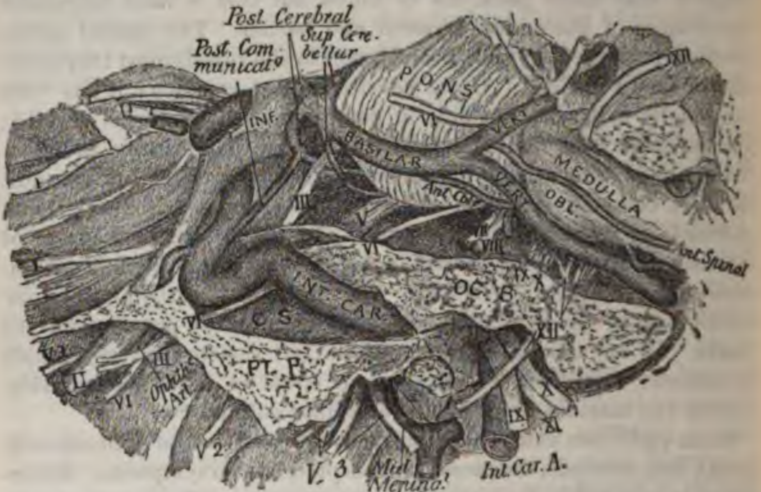

FIG. 143.—Relations of the arteries and nerves at the base of the brain, from an actual dissection in which the bones, membranes, and wall of the cavernous sinus were removed from the left side. INF, infundibulum; C. S., cavernous sinus; Pt. P., pterygoid process; OC. B., occipital bone. The cranial nerves are indicated by Roman numerals; the names of the arteries are marked upon them. (After a drawing, by Mr. C. E. Adams, of Mr. Shattock's dissection.)

Anterior Cerebral.—The aneurism is usually in front of the origin of the vessel, sometimes at a bifurcation. The artery runs between the optic and the olfactory nerves, and an aneurism here exerts compression on the same parts, and produces nearly the same symptoms, as one of the internal carotid, with the important exception that the nerves to the muscles of the eyeball generally escape. An aneurism further forwards may cause no local symptoms, but when the blood-supply to the cortex is interfered with, hemiplegia results, and aphasia, if the disease is on the left side. The sac usually has very thin walls, and rupture occurs in more than half the cases, often comparatively early. The blood has been known to find its way to the lateral ventricle.*

Anterior Communicating.—The tumour exerts pressure only on the inner surface of the frontal lobes. The aneurism is usually small, and, as a rule, no symptoms are produced. The wall is thin, and here also rupture occurs early in most cases.

Posterior Communicating.—The aneurism is usually situated near the origin of the artery from the internal carotid. It compresses the adjacent temporo-sphenoidal lobe, and the pressure may even reach the corpus striatum and optic thalamus. The third nerve suffers most frequently. Affection of sight is also common, but it is usually incomplete, and we have no exact observations on its character. Probably there is hemianopia from pressure on the optic tract, which the artery crosses. Hemiplegia is rare, in spite of the proximity of the vessel to the crus cerebri, because the aneurism is thin-walled, and usually bursts before it has attained considerable size, or has exerted considerable pressure.

Middle Cerebral.—The aneurism is sometimes near the origin of the vessel, but more often within the fissure of Sylvius, sometimes on one of its cortical branches. Occasionally there is more than one dilatation. The chief pressure is on the hemisphere, and when the aneurism is within the fissure of Sylvius the pressure may reach the corpus striatum. Paralysis of cranial nerves is rare; on the other hand, hemiplegia and convulsions are common. Speech is occasionally affected when the disease is on the left side. Rupture occurs in two thirds of the cases. Embolism is the common cause, and if the inflammation engendered involves the adjacent membranes, these may be so thickened as to resist external rupture. As mentioned, blood, under these circumstances, may work its way, in the course of a few days, from the convexity to the lateral ventricle. Aneurisms on the central branches of the middle cerebral are small, and probably cause no symptoms until they burst.

Vertebral.—A distinct aneurism, such as is shown in Fig. 106, p. 250, is extremely rare, although both arteries have been thus affected (see p. 425). But in most conditions described as aneurism there has been only a general dilatation of the vessel, often determined by an original

* Mott, 'Brain,' 1889.

inequality in the size of the two arteries. Important as are the adjacent structures, symptoms have seldom been observed in the cases in which such dilatation was found. In the case figured (p. 250), facial spasm was the result of the compression of the trunk of the facial nerve.

Basilar.—While absolutely second in frequency of affection, if the shortness of the vessel is taken into consideration, the basilar exhibits a greater tendency to suffer than any other artery; this is chiefly due to its frequent affection by syphilitic disease. Occasionally there is general dilatation; more frequently the anterior extremity is enlarged; sometimes there is an aneurism on one side of the vessel in the middle of its course, rarely near the origin from the vertebrals. The pressure is differently distributed according to the position of the aneurism. Usually the pons suffers chiefly, sometimes on one side only; the pressure may even extend to the middle peduncle of the cerebellum and the adjacent cerebellar hemisphere, or so deeply as to reach the fourth ventricle. The compressed portion is often softened. An aneurism of the anterior part may compress the crus; one of the posterior part, the pyramids and olivary bodies of the medulla. The third nerves rarely suffer, but the cranial nerves, from the fifth to the vagus, are frequently compressed; the hypoglossal usually escapes. Internal hydrocephalus is an occasional consequence of the obstruction to the passage of liquid from the ventricles. Rupture occurs in about half the cases; it is usually subarachnoid, although the blood sometimes escapes into the substance of the pons. It is remarkable that in about one third of the cases there have been no symptoms of the tumour; when present they have consisted in occipital headache, vertigo, and the symptoms common to basal tumours in this situation—paralysis of the limbs, bilateral or unilateral, and of one or more cranial nerves on the opposite side. The fifth suffers most frequently, and sometimes there is neuro-paralytic ophthalmia. Difficulty in articulation and in swallowing are occasional symptoms. Convulsions are rare. Besides the terminal apoplexy which results from rupture, apoplectic attacks occasionally occur, and even end in death, without rupture, in consequence of softening adjacent to the aneurism, which is sometimes due to the closure of a branch.

Posterior Cerebral.—The aneurism is usually situated not far from the origin of the vessel, and may compress the upper part of pons, crus, temporo-sphenoidal lobe, and the third and sixth nerves. Hence, when symptoms have existed, there has been hemiplegia with alternate paralysis of these two nerves, especially of the third.

Cerebellar Arteries.—Both the superior and inferior anterior cerebellar arteries have been found affected with small aneurisms, but the pressure-effects are too various, and usually also too slight, to be of practical importance.

Course.—The duration of an aneurism can only be inferred from the inadequate evidence afforded by the duration of the symptoms.

It is certain, however, that this varies between wide limits, from a few weeks to five or six years. In some cases the sac becomes filled with clot, and a spontaneous cure is thus effected.* The majority of cases have ended by the rupture of the aneurism, which is, so far as past experience has gone, invariably fatal. Quickly deepening coma, and sometimes convulsions, result from the hæmorrhage into the membranes, and death often results in a few hours, although sometimes the hæmorrhage occurs so slowly that life is prolonged for days. Now and then one or more remissions occur, the cause of which can be traced, post mortem, to be the temporary cessation of the hæmorrhage, the blood having evidently been effused at different dates. Thus one patient lived for fourteen days after the first rupture of an aneurism of the anterior communicating artery. When the hæmorrhage occurs into the brain-substance, the symptoms are similar to those in ordinary cerebral hæmorrhage.

Diagnosis.—In a considerable number of cases the diagnosis of intra-cranial aneurism is impossible, because, until its rupture, it produces no effects by which its presence can be recognised. When symptoms are present, they are commonly those of a tumour, generally at the base of the brain. The aneurism *is* a tumour, and its distinction from a growth is often impossible, and is only quite certain when a murmur exists in it. But such a murmur has only been heard in a few cases of aneurism of the internal carotid or vertebral arteries. The distinction may, however, be made with probability when the symptoms of a tumour, in the position of a vessel, develop in connection with one of the causes of aneurism, especially heart disease. Syphilis and injury have less diagnostic significance, because each (and syphilis especially) is a cause of growth more frequently than of aneurism, but this significance varies according to the seat of the disease. In the cavernous sinus, for instance, an aneurism is far more probable than a growth. Moreover, in cases of syphilis, the fact that treatment fails to exert an influence on the tumour is often strong evidence of its aneurismal nature.

Whatever be the apparent cause, it is of great importance to recognise the correspondence in the position of the tumour with that of one of the arteries liable to be thus affected, and to remember the special relation of some causes to certain arteries, as already described, as embolism to the middle cerebral and syphilis to the basilar.

The evidence of the existence and seat of an aneurism have thus to be frequently considered together, but the latter is not often unequivocal. The seat of pain is of significance only when it is occipital (basilar artery). Loss of sight in one eye, sometimes extending to the other—with, or especially without, optic neuritis; with or without loss of smell on the side first affected,—occurs in aneurism of the internal carotid or anterior cerebral; and the distinction between the two depends on the presence or absence of paralysis of the other nerves to the eyeball first affected, which occurs especially when the aneurism is of the internal carotid. Paralysis of the third nerve, without loss of sight in one eye, is produced by aneurism of the posterior communicating artery, and, with hemiplegia on the opposite side, by aneurism of the posterior cerebral. Hemianopia would increase the probability of an aneurism in this part of the base. Affection of the fifth alone is of little significance, but with bilateral weakness in the limbs, difficulty of articulation or of swallowing, it is produced by aneurism of the basilar, and so also is paralysis of the cranial nerves below the sixth, usually associated with weakness of the limbs on the opposite side. Hemiplegia is of little significance in itself, but if considerable it is unlikely that there is an aneurism of either the anterior cerebral or the posterior communicating arteries. General convulsions, epileptiform in character, are not common, but if they begin locally they suggest that the disease is in the middle cerebral, and if they are opisthotonic, that it is in the basilar. These symptoms are of diagnostic significance only in the presence of a cause of aneurism.

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* A case in which this occurred in an aneurism of the internal carotid has been recorded by Mr. Hutchinson ('Clin. Soc. Trans.,' 1875).

The occurrence of rupture, with severe apoplectic symptoms, increases very much the probability of the diagnosis, although it is then a matter rather of scientific curiosity than of practical importance. Even if no previous symptoms have existed, it may be suspected that sudden apoplexy, steadily deepening in degree, is due to the rupture of an aneurism, if it occurs in a young person, or if, before the degenerative period of life is reached, there is a history of heart disease or of syphilis, or of injury to the head. The characters of the apoplexy afford but limited indications of the probable seat of the aneurism. Those suggestive of meningeal hæmorrhage are of no localising significance, but those of hæmorrhage into the pons suggest an aneurism of the basilar, or of the commencement of the posterior cerebral artery.

The difficulties of the diagnosis of aneurism do not always cease with life. The damage to the tissues and vessels, caused by the hæmorrhage that follows rupture, may render it impossible to find the remains of a small aneurism, especially of one with thin walls, difficult to discern in the mass of clot around it. But meningeal hæmorrhage, under the conditions that suggest aneurism, is enough to justify a strong suspicion of its existence.

Prognosis.—If the diagnosis of an intra-cranial aneurism is certain, the prognosis is extremely grave. The probability that death will result is very great, and the duration of life is most uncertain, since it is impossible to say how near or how distant may be the fatal rupture. These conclusions are not invalidated by the fact that, in a considerable proportion of recorded cases, rupture has not occurred. In most of these the aneurisms were small, and caused no symptoms. The question of prognosis relates only to aneurisms which have caused symptoms, and in most of these cases rupture has occurred. But the end may not be near; in many cases the symptoms have continued for two, three, or five years, and if stationary the chance of long duration is slightly greater. Nor is a fatal issue absolutely certain. A spontaneous cure of intra-cranial aneurism has been more than once observed. In aneurism of the internal carotid the prognosis is distinctly better than in that of other arteries, because more can be done by treatment, or at least has been done, than when other vessels are the seat of the disease.

Treatment.—If an intra-cranial aneurism is suspected, it is important to avoid as far as possible all influences which tend to increase the arterial pressure, as exertion and stooping, or which tend to accelerate the movement of the blood, as alcohol. To these ends also the bowels should be kept loose, and the patient should sleep with head and shoulders well raised. If there is reason to suspect that the aneurism is of syphilitic origin, iodide of potassium should be given to prevent increase of arterial disease elsewhere; although it is not to be anticipated that it can influence an aneurism already developed, it may promote coagulation in it. Ergotine has been recommended, but the wall of an aneurism contains no tissue on which the drug can act. More reasonable measures are those designed to favour coagulation in the sac. These consist, first, in the "starvation" treatment, the utility of which in intra-cranial aneurism has still to be proved, whatever be the origin of the aneurism. Secondly, iodide of potassium has been given to promote coagulation, and with a result which certainly warrants its employment. In one case* the patient, a woman aged forty, suffered from right frontal headache, photophobia, impaired vision in the right eye, and paralysis of the external rectus; a systolic murmur could be heard, loudest at the right temple. Iodide of potassium was given in doses increasing to 36 grs. daily. Four months later, after a violent attack of vomiting and purging, the murmur suddenly ceased, and did not recur. In five weeks the paralysis of the external rectus had disappeared. There can be no doubt that an aneurism existed, and that coagulation had occurred in the sac. The immediate cause of the clotting was probably the prostration from the vomiting and purging, but it is at least possible that the result may have been favoured by the iodide of potassium, since this drug certainly promotes coagulation in aortic aneurisms. The third method of promoting coagulation in the sac is to ligature the artery from which the diseased vessel derives its supply of blood. Hitherto this has only been adopted in the case of aneurism of the internal carotid, for which the common carotid has been tied with success. It is only in aneurism of this artery that the diagnosis has been sufficiently sure to justify the surgeon's interference. If other aneurisms could be diagnosed with equal certainty, this measure would be more often justifiable. For basilar aneurism, the vertebrals would have to be tied, but Dr. Alexander, of Liverpool, has shown that this operation is practicable, and, if not devoid of danger, is attended with less risk than is the aneurism itself. When rupture has occurred, no treatment has hitherto had any influence, but the onset of apoplexy increases the indication for ligature, and likewise its justification, unless the end is obviously too near to permit life to be saved.

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* Recorded by Dr. Humble, of Corfe Castle, 'Lancet,' 1875, ii, pp. 490 and 874.

DEGENERATIONS OF THE BRAIN.

CYSTIC DEGENERATION.

THE shrunken brain of the old is occasionally riddled with small cavities, from the size of a pea downwards. The change is seen chiefly in the white substance, and is sometimes general, sometimes limited to certain parts. The cavities are the enlarged perivascular spaces, and the alteration seems to represent, in the substance of the brain, the senile atrophy which causes the surface to shrink, the sulci to be widened, and the subarachnoid fluid in them to be increased in quantity. It is also usually associated with a similar enlargement of the ventricles. As Charcot has pointed out, the most frequent seat of this cystic degeneration is a zone of the white substance between the region supplied by the cortical arteries and that of the central vessels, in which the terminal branches of each set are inadequate for the perfect maintenance of nutrition. It is not probable that these cavities, however numerous, give rise to symptoms, any more than does the general shrinkage of the brain, but the arteries within them have but little support, and dilatation and rupture may be facilitated, especially when the cavities exist outside the lenticular nucleus, where cerebral hæmorrhage so often takes place. They often contain *débris* of nerve-tissue, and hæmatoidin crystals, the remains of some small extravasation. Occasionally, especially in the pons, the cavities are met with at an earlier age, even in young adults, and are then sometimes thought to have a pathological significance which they do not really possess.

CHRONIC PROGRESSIVE SOFTENING.

The old view, that softening may occur as a primary process, although wrong as regards the common acute softening, seems to be justified by rare cases in which softening occurs without demonstrable association with disease of the arteries, or correspondence with their distribution, and is so gradual in its onset, and slowly progressive in its course, as to make a dependence on the vessels very improbable. Some cases of this kind have been collected by Wernicke,* and a few other instances are to be found in medical literature, old and recent. The affection is almost always seated in the white substance of the hemisphere, a feature in which it differs from the acute softening of actual obstruction. But there is no reason to suppose that the grey matter invariably escapes. The change consists of simple white softening, which presents, under the microscope, chiefly the products of degeneration. The affected area is often sharply limited from the adjacent normal substance. The extent of the lesion varies much in different cases: subcortical softening has even extended through the whole convexity of one hemisphere. Although the grey matter of the cortex has not been primarily affected in the cases examined, it may suffer secondarily when the softening of the white substance is extensive. The diffluent tissue is then limited externally by the pia mater, and sometimes there is a thin layer of cortical grey matter below this. Chronic softening may also affect the cerebellum, but it there differs from the cerebral softening, since the cortex suffers more than the white substance. I have seen the cortex of almost the whole of both hemispheres of the cerebellum uniformly softened and atrophied, while the white substance was unaffected. In this case there were also several areas of softening in the cerebral hemispheres, in the white substance and in the grey matter of the cerebral ganglia.

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* 'Lehrbuch der Gehirn-Krankheiten,' Bd. ii, p. 149.

The recorded cases are still too few to afford much information regarding the causes of chronic progressive softening. All that can be said is that it occurs in both sexes, and is a disease of the second half of life, most frequent between sixty and eighty. The chief symptoms are hemiplegia—motor and sensory—of gradual onset. Weakness commences in one part and gradually spreads to the whole of one side, but without considerable sensory loss. Slight irritative symptoms occur, numbness, tingling, and formication in the limbs, and in some cases these have been the seat of paroxysmal pain. Rigidity may accompany the commencing paralysis, but ceases when it is fully developed. Convulsions are very rare. There may be occasional attacks of giddiness, but apoplectic seizures do not occur in the chronic form. The intellect may be little affected, or the patient may become dull and apathetic, and, towards the last, comatose. But the cerebral symptoms have usually reached a certain degree of severity, and then remained stationary. Death ultimately occurs from bedsores, pneumonia, or some intercurrent senile malady, perhaps favoured by the nervous state. The duration of recorded cases has varied between a few months and two years, but the onset cannot easily be fixed.

Although it is not probable that the condition is directly related to arterial disease, the latter is present in most senile cases, and may have an indirect influence in determining the lesion, its common seat, the white substance, being nourished partly by the central, partly by the cortical vessels, with the effect just mentioned in the account of cystic degeneration. Subacute and acute cases are met with, which present every gradation of course between the chronic form, the less rapid forms of softening from arterial occlusion, and focal inflammatory softening. It is especially important to keep in view the cases, in the old, in which the symptoms suggest a focal chronic inflammation (mentioned on p. 469). Pathological facts are urgently needed regarding all these forms, and the subject is commended to the notice of those under whose observation, in workhouses for instance, the diseases of the old so often come.

It will be seen that the chronic symptoms bear a considerable resemblance to those of cerebral tumour. A difference is in the fact that headache is more often absent than present, and very rarely reaches the degree of intensity that is so characteristic of tumour. Optic neuritis has also been absent in the cases in which the eyes have been examined. The greatest diagnostic difficulty is presented by cases of very chronic course. If symptoms develop in a subacute manner, reach a considerable degree of intensity in six weeks or two months, and then remain stationary, the distinction from tumour is more easy, because a rapidly growing new formation rarely undergoes early arrest. The age of the patient is also of some significance; in the first half of life, tumour is more probable than softening. The occurrence of general convulsions makes softening unlikely, but cortical fits may be produced by softening situated at the motor region.

Little can be said regarding the treatment of these cases. Our knowledge of them depends only on the cases in which the nature of the disease has been ascertained by a post-mortem examination, and in these it does not appear that any treatment employed has had an appreciable influence on the course of the disease. The most suitable measures will probably be those recommended for the chronic stage of acute softening—rest, nutritious food, with general and cardiac tonics.

DISSEMINATED OR INSULAR SCLEROSIS.

The disease thus named consists in the formation of scattered islets of sclerosis in various parts of the brain and spinal cord, sometimes also in the cranial nerves. Our knowledge of the affection is recent. The lesion was, indeed, long since figured by Carswell and Cruveilhier, and it was described, thirty years ago, by Frerichs, Rindfleisch, &c., but the malady was not generally recognised until re-investigated by Vulpian, and by Charcot and his pupils, and described by Charcot in his widely circulated 'Lectures.' It is called by the French "Sclérose en Plaques Disseminées," by the Germans "Multiple Sclerosis," and, in this country, the French designation has been modified into "Disseminated Sclerosis;" but it is also called "Insular Sclerosis," a convenient term, proposed by the late Dr. Moxon.

The pathological relations of the morbid process are still, to a large extent, obscure, and much remains to be done in elucidating its clinical history and pathological relations. As regards the symptoms of the typical form, little can be added to the description given by Charcot, but there is much uncertainty regarding the untypical varieties, which are by no means rare.

Three forms of the disease have been distinguished—cerebral, spinal, and cerebro-spinal, according to the situation of the islets of sclerosis, and the corresponding difference in the symptoms they produce. The cerebro-spinal variety must be regarded as the typical form, which gives rise to the complete series of symptoms; but the disease may be at first limited to one part, and may afterwards become general; or it may remain limited, so far as symptoms are concerned, to the part first affected. Hence the nature of many cases is misunderstood, and other diseases have often been described as examples of this affection; its name having become familiar, it is apt to be suspected where it does not exist.

Etiology.—Insular sclerosis occurs in both sexes with nearly the same frequency. It is met with at almost all periods of life between childhood and old age, but especially in the first half of adult life. The majority of the cases commence between twenty and thirty-five. In one verified case it commenced at seven, and its symptoms have been observed still earlier; in another case it began as late as sixty, but in old age it is extremely rare. Direct heredity, or the affection of two brothers or sisters, has been noted in a few instances, but is quite exceptional; indirect inheritance is more common, shown by a family history of insanity, epilepsy, or of some form of chronic paralysis. Often, however, no hereditary predisposition can be traced. In about half the cases the disease develops without any recognisable cause, near or distant. Among the influences to which it has been immediately ascribed, the most frequent are exposure to cold, mental distress, over-exertion, some acute disease, an actual lesion of the central nervous system of traumatic or other nature, or some specific febrile disease. But any single exciting influence is to be traced only in a very small proportion of the cases, and the most noteworthy fact regarding them is the occurrence of the disease at a time of life when general influences have, as a rule, no late sequelæ. Among the acute diseases that have preceded it, typhoid fever and smallpox are the most frequent; it has also been known to follow diphtheria and erysipelas. The preceding morbid processes in the nervous system have been apparently acute or subacute inflammation (myelitis, &c.), primary, or secondary to some injury. Their symptoms have subsided, or have left some stationary residual effects; after an interval of a year or more, the indications of insular sclerosis have slowly developed. I have known it to begin during pregnancy, remain stationary until the next pregnancy, and then become progressive.

Preceding syphilis can scarcely ever be traced, so that it is doubtful whether the very rare cases in which symptoms of this disorder in syphilitic subjects have been removed by treatment were really due to sclerosis.[*]

Pathological Anatomy.—The islets of sclerosis that characterise the disease are always irregular in situation, scattered, as it were at random, through the central nervous system, subject only to the most general laws of distribution, and corresponding in locality in no two cases. The only law which can be traced is that they generally commence in the white substance. In the brain they seldom invade the grey matter of the cortex, and never seem to begin in it, however numerous they may be in the white substance of the centrum ovale, a favourite seat. They are irregular in shape, and vary in size from that of a pea (or less) to that of a walnut. In colour they are reddish grey, a little darker than the normal grey substance of the cortex, and rather more translucent. Their section is usually on the level of that of the adjacent cerebral tissue; occasionally it is a little depressed, but on

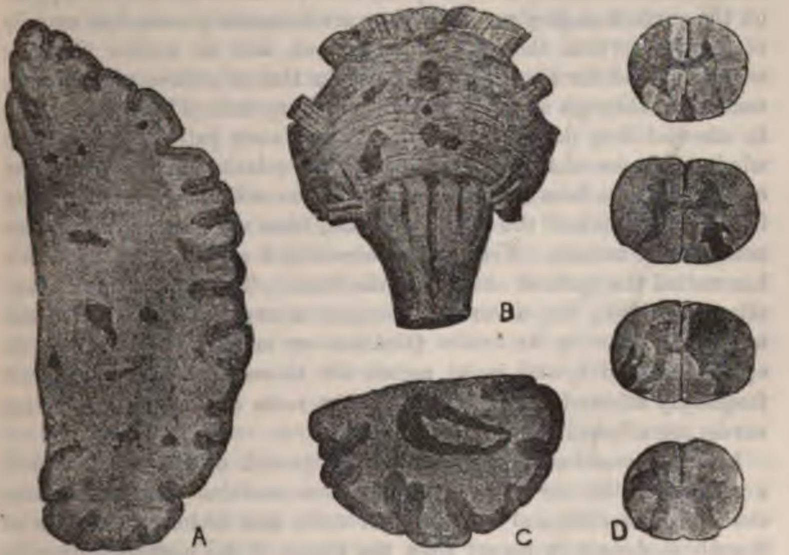

Fig. 144.—Insular sclerosis.

A. Centrum ovale of right hemisphere.

B. Pons and medulla.

C. Peri-ependymal sclerosis around the descending cornu of the lateral ventricle. (A, B, and C are from an original case.)

D. Sections of the spinal cord to show the varying distribution of the sclerosis at different regions. (After Leyden.)

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[*] Some facts bearing on this question will be considered in the section on "Pathology."

the surface (as in the crus or pons or cord) they are sometimes slightly prominent. The sclerosis does not, however, increase perceptibly the volume of the part in which it occurs. To the naked eye the islets are sharply defined; the transition to the normal tissue is sudden. The consistence of the sclerosis is usually a little firmer than that of the brain-tissue, and they are sometimes distinctly firm; occasionally, in old cases, some may be almost cartilaginous in their firmness. Owing to the position of the islets in the cerebral hemisphere there is usually no indication of their presence on the surface of the brain, even when a large number are found in its interior. Although the cortex is seldom involved, the central ganglia are frequently invaded, and the sclerosis is sometimes abundant beneath the lining membrane of the lateral ventricle (Fig. 144, C) and in the corpus callosum. In the crus and pons the islets are often numerous; some may be conspicuous on the surface from their contrast with the white fibres which constitute the superficial layer of these parts, while others are scattered through the interior of the pons or medulla. They are met with also in the cerebellum, but are usually few, and here also are confined to the white substance. In the spinal cord the areas of disease appear on the surface as grey patches; they are irregular in size, but usually of greater vertical than transverse extent, and on section they are seen to extend for a variable distance in the substance of the cord, sometimes through a considerable part of one half (Fig. 144, D), and, in cases of long duration, the disease may even extend through the whole thickness of the cord. Such diffuse sclerosis of the lower part of the cord has been met with in association with characteristic islets in the upper part of the cord and brain, from which it probably does not differ in nature. Very rarely, however, a slight diffuse sclerosis has united the spots of disease in the brain. The cranial nerves are often involved; for a certain distance a nerve may be grey and sclerosed either in its entire thickness or in part. The olfactory, optic, third, fifth, and facial nerves are those that have been most frequently affected. Less commonly the roots of some of the spinal nerves are affected.

In the diseased areas there is an overgrowth of the neuroglia and a wasting of the nerve-fibres. The tissue contains connective-tissue elements, glia-cells and fibres, spider-cells, and ultimately a mass of fine fibres, denser in aspect than the fibres of the normal neuroglia. Among these tissue-elements there may be fatty granules and globules, the remains of the degenerated nerve-elements. The nerve-fibres that pass through are changed in various degrees. The chief alteration is a narrowing of the white substance, which becomes reduced (as seen in section) to a narrow, scarcely appreciable ring around the axis-cylinder, and even this may cease to be recognisable. The axis-cylinders persist longest, but ultimately they may disappear.* The walls of the vessels become thickened, and the increase of the adjacent connective tissue may be very conspicuous. Should the sclerosis involve the grey substance, the nerve-cells become atrophied, and the intervening structures suffer changes like those of the white substance, but, from its nature, less readily perceived. Over affected areas that reach the surface there may be a slight thickening of the pia mater, but seldom sufficient to be recognised with the naked eye.

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* The persistence of the axis-cylinders was first pointed out by Charcot, and has been since confirmed by Schultze, Déjérine, Babinski, and others.

If an area of sclerosis occupies part of a tract of fibres that is liable to undergo secondary degeneration, such degeneration may or may not be found beyond the part diseased, but it is usually present in some degree. The difference seems to depend on the condition of the axis-cylinders; as long as the change is confined to the white substance of the nerve-fibres there is no secondary degeneration, but as soon as the axis-cylinders suffer, degeneration occurs.

When the optic nerve undergoes general atrophy, the same tendency is seen for the axis-cylinders to persist and the medullary sheath to suffer. The connective tissue is much increased between the bundles and in the inner nerve-sheath, and the nuclei between the nerve-fibres are multiplied (Uhthoff). An islet of sclerosis in this or any nerve presents the conditions already described.

SYMPTOMS.—The wide variations in the position of the islets of sclerosis entails a corresponding difference in the symptoms that are produced. Loss of muscular power in the limbs, variously distributed, and often at first partial, is the most constant manifestation of the disease. Since the morbid process usually involves the pons and spinal cord as well as the brain, some islets can hardly fail to occupy the motor tract. The deficiency of power in the arms is very variable in degree, but the characteristic feature of the weakness is its association with a peculiar symptom, jerky irregularity of movement, wide in range, and ceasing on rest. It may exist also in the neck muscles, and in the legs; also in the latter it is often masked by the general unsteadiness it causes, but it is sometimes conspicuous, even when the arms are little affected; ataxy may exist alone, but simple spasmodic paraplegia is still more common. Some of the cranial nerves are often also paralysed. Among other symptoms, nystagmus is exceedingly common, and so is a peculiar disturbance of articulation. These symptoms vary in occurrence and degree, in different cases, to an extent which makes it impossible to give a description of the disease alike accurate and comprehensive, and also gives rise to numerous difficulties in its diagnosis.

The variation in the character of the symptoms extends to their order and to their mode of onset. In some cases the peculiar inco-ordination in the hands, or in one alone, is the first indication of the disease. In other and more frequent cases the legs first become weak, or the patient is unsteady in standing or walking. Dull pains in the limbs may trouble the patient for a year or two before the onset, or a sense of "numbness" in the hands and feet, occasionally with actual lessened sensibility, may occur and pass away, or even recur more than once. Less commonly some cranial nerve-palsy occurs at the onset, or speech is observed to be peculiar, but in the majority of cases the limb symptoms precede the others. It is important to remember that some of the preceding symptoms may be due to a morbid process connected with the cause of the disease.

Of the individual symptoms, the jerky irregularity or tremor is the most peculiar and characteristic. It is, as just observed, manifested most frequently in the arms. At rest the limb is still, and presents no spontaneous tremor, but as soon as a movement is attempted, in the endeavour, for instance, to take hold of some object, the arm is thrown about in a wildly irregular manner, and moves in sudden quick jerks, now in one direction, now in another, until at last by a great effort, and sometimes with a sudden dash, the patient succeeds in grasping the object. If he tries to raise a glass of water, the movements are so wild that the contents of the glass are probably spilled. In an attempt to write, the violent movements produce only irregular strokes upon the paper as the hand dashes to and fro, and often the paper is pierced or even the pen is broken in the attempt. In some instances the movements are slighter in range and more regular, even resembling simple tremor, although its relation to the malady may be proved by the presence of the typical form in some other part. It is said that in rare instances the movements have been known to continue during rest, but there is some doubt as to the nature of such exceptional cases. The movements are, as a rule, increased by effort, by motion, and by attention. The more the patient tries to overcome them, the more violent they are, but the variations in degree are not great, and the disorder of movement is unceasing.*

The muscles of the neck often share the disorder of movement, and there is irregular oscillation of the head, and sometimes of the trunk, whenever the patient sits up. The derangement may even be greater in the muscles that support the head than elsewhere, and it may cause so much unsteadiness that the head has to be kept still by the hand to permit the patient to drink. When laid at rest the head at once becomes still. Although the affection of the movement of the legs usually causes unsteadiness, which may render walking impossible without assistance, and even standing difficult, they sometimes present the irregularity of movement above described. It will be seen that the distribution of this symptom varies much, and with it the general aspect of the cases. For instance, while it is usually most marked in the arms, one unquestionable case presented in these only the fine irregular tremor on sustained movement, great inco-ordination in the legs, and extreme jerky disorder in the muscles supporting the head. Rarely the limb symptoms exist on one side, alone or chiefly; one limb is then more affected than the other, as in a case in which the left leg was merely weak, while the left arm presented typical irregularity of movement. Such instances might be multiplied indefinitely; they are explained by the great variety in the lesions.

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* Hence they are called by the Germans "Intentions-Zittern," a term which cannot be rendered in English without inaccuracy, on account of the meaning of "intention." "Movement-tremor" embraces all varieties, but fails to indicate the irregularity of the typical form. Perhaps "atactic tremor" would be, on the whole, the best term, because there is no ataxy without voluntary movement.

Some weakness usually accompanies this irregular inco-ordination; it may be at first slight, and indeed, at the very onset, it may be absent. On the other hand, weakness may exist alone, especially in the legs. Once established, the loss of power gradually increases.

The jerky irregularity has been ascribed by Charcot to an irregular resistance to conduction in the nerve-fibres at the sclerosed spot; it is supposed that the changes in the white substance, or in the axis-cylinder, interfere with conduction along the latter, and as this change is unequal in degree, even in adjacent fibres, there is a various degree of retardation of the voluntary influence on the muscles, and hence the irregularity of the contractions. This hypothesis derives some support from the fact that a precisely similar symptom is present in some cases of tumour of the brain, in which, for instance, a tubercular growth compresses the motor path, as in the case of tubercle in the crus shown at Fig. 142, p. 509. I have met with this symptom also in a case of tubercle of the pons. To some (Erb, &c.) the theory has appeared inadequate, and they incline to the alternative explanation, that the disorder is due to the occurrence of islets of sclerosis in some particular situation in which they derange the co-ordinating function. The symptom is said to coincide with sclerosis of the pons, and to be absent in cases in which the lesion is confined to the spinal cord.

Simple weakness in the legs is common, and is often the earliest symptom of the disease, and may present a paroxysmal increase on exertion, so that the patient falls occasionally, and can only resume walking after a rest. When it is considerable it is usually accompanied by an excess of myotatic irritability (increased knee-jerk, foot-clonus, &c.), which goes on to spasm, so that a state of spastic paraplegia is established. This is indicative, as in other cases, of degeneration of the lower parts of the pyramidal tracts. Such degeneration in this disease is secondary, the consequence of the development of areas of sclerosis somewhere in the dorsal region of the cord, involving the pyramidal tracts. In the rare cases in which the lesion is limited to the spinal cord, such spastic paraplegia may be the only symptom of its presence, and many cases have been mistaken for primary lateral sclerosis. The ataxy of the legs, present in other cases, varies in its character, and probably also in its mechanism. There may be, as we have seen, jerky inco-ordination with which the myotatic irritability is usually increased. In other cases there is simple unsteadiness, sometimes like cerebellar, sometimes like spinal inco-ordination. It is probable that this is due to the interruption of the upward paths from the muscles, as explained in vol. i. In a rare group of cases the state of the legs is identical with that in true tabes, perhaps in consequence of a patch of sclerosis in the posterior columns in the lumbar region; but it is also possible that the condition is true tabes, a system-degeneration in one part of the central nervous system accompanying the insular sclerosis in another part. This association will be considered in the section on pathology.

The excess of the muscle-reflex action in the legs may increase to rigidity, the common variable extensor spasm, or, in rare cases, flexor contracture, so that the legs are permanently drawn up. Occasionally a single group of muscles may become rigid and contracted in the arms as well as in the legs. Muscular wasting is rare, but local irregular atrophy may result from the invasion of the grey matter of the cord by some islet of sclerosis.

Cutaneous sensibility is normal in the early stage of the disease, but it may afterwards be impaired. The loss varies much in its distribution according to the situation of the sclerosis on which it depends. Rarely there is hemianæsthesia from the development of an islet in the sensory path within the brain. The loss is more often partial, involving only a small area on a limb, and it may or may not correspond to the motor palsy. When there is diffuse sclerosis of the cord there may be extensive loss of sensibility on the legs. The condition of sensation is intelligible when we remember how slowly the axis-cylinders of the nerve-fibres suffer, and that a greater amount of damage is required to abolish sensory conduction than suffices to cause motor palsy. Irritative symptoms are occasionally present; a sense of vague numbness or formication in the hands, sometimes also in the feet, may be a very early symptom, and may even pass away before other disturbance is developed, although it occasionally persists. Dull aching pains in the limbs are not uncommon, especially in the legs, sometimes in the back, and may occur from time to time throughout the course of the disease. Sharp momentary pains also occur, but not often. A girdle pain may occur, early or late, persistent or transient, complete or partial, but also is not frequent. These sensations are chiefly complained of when there is other evidence of the presence of sclerotic islets in the spinal cord, but even then they may be absent, and the comfort of the patient may be undisturbed.

Eye symptoms are frequent and important. The optic nerve may suffer in various ways. There may be impairment of sight in one eye or in both, often with an irregular contraction in the field of vision, and without at first any visible changes in the optic nerve to account for it. This condition depends upon the development of an islet of sclerosis in one or both optic nerves, or in the optic chiasma. It may progress to almost complete blindness of one eye. After a time, secondary atrophy supervenes, and can be seen with the ophthalmoscope. Occasionally the atrophy is preceded by slight neuritis, when the sclerosis is near the eyeball. In some cases, again, there is a primary atrophy of the optic nerves, exactly like that which occurs in tabes, with a similar failure of sight, proportioned to the visible alteration in the nerve. Both eyes usually suffer, but one is often affected earlier and more than the other. This complication is, according to my own experience, rather more frequent than the form first described, and is of much pathological interest.

The internal ocular muscles are seldom involved, but each form of internal palsy is sometimes met with, loss of accommodation rather more frequently than loss of the light-reflex alone. The pupils are sometimes unequal, or become so on accommodation.

On the other hand, the external muscles often present some abnormality. The most frequent disturbance is nystagmus, which is an exceedingly common symptom of the disease, although we cannot explain it. Both forms occur, spontaneous and on movement, but the former is uncommon; the latter is that usually present, and it does not differ in features from that which is met with in other central degenerations. It is generally on lateral or upward, seldom on downward rotation. The quick displacement of the globes is, as a rule, in the direction of the voluntary movement, but curious variations are sometimes met with, especially in association with loss of power. One patient could only move the eyes to the left halfway to the canthus for a moment, but nystagmus only occurred on movement to the right. In another case, on movement to the right there was nystagmus with the quick movement to the right; on movement upwards there was rotatory nystagmus, and vertical oscillation when the eyes were directed to the left. In another the quick motion was to the right, whether the movement of the eyes was towards the right or the left side. These variations quite baffle our power of explanation.

The external palsies are more often of a conjugate movement than of a single nerve or muscle, except the external recti (sixth nerves), which may be weakened on one or both sides; one superior rectus is occasionally feeble. But convergence, and the movement of both eyes towards one side, are the motions most often impaired, and they are occasionally lost. In exceptional cases, however, there is considerable and increasing palsy of one of the ocular nerves, as the third, due to the development of sclerosis in the nerve-trunk. Transient diplopia is also occasionally complained of, even in the early stage of the disease.

Other cranial nerves may also become paralysed by the same mechanism and with the same rarity, especially the facial, fifth, or hypoglossal nerves. In the affection of the first of these, all parts of the face suffer, and the degenerative reaction is well marked. Very rarely there is clonic spasm in the face, which may even be the first symptom. In the fifth nerve, either one or both parts may be involved. The implication of the sensory part causes much less pain than is produced by most other lesions of this nerve-trunk. The sclerosis that thus damages these nerves may be either in the nerve itself, near the surface of the brain, or in the pons or medulla, at the central course of the fibres. Difficulty in swallowing may be present when the pons or medulla is the seat of the morbid process.

Another very frequent symptom—so common as to be of diagnostic importance—is a peculiar change in articulation. Syllables (less frequently words) are unduly separated and unduly accentuated, in what has been termed "staccato," or "syllabic," or "scanning" utterance, often with a tendency to elide the ends of words. Some patients, indeed, present only elisive or slurring articulation. The syllabic form suggests a difficulty in uttering the syllables, and there may be an actual consciousness of this difficulty. The movement of the tongue is sometimes jerky and irregular, but the face is seldom involved, and the peculiar affection of speech is independent of any visible disorder of the movement of the tongue. It has been met with when the pons and medulla were free from sclerosis, and its source is thus obscure.

Slight mental change is common; considerable alteration is very rare. There may be failure of memory, but especially frequent is an undue complacency and contentment, which, under the increasing disability, is distinctly unnatural. There is not often any mistaken idea of improvement, but the patient may seize on and exaggerate any distinct diminution in the symptoms. This morbid complacency is rather more frequent in women than in men, and in young adults than in later life. Hysterical and emotional disturbance may also occur, even in men. Very rarely mental disturbance is considerable enough to amount to chronic insanity.

Among other symptoms that are frequently met with are headache and giddiness; both may occur early in the disease, and are usually paroxysmal. The vertigo is especially pronounced, but its frequency has been over-estimated, and after a time it ceases to occur. The attacks may be very severe and come on apart from any apparent cause. As in other forms of severe giddiness, it may be accompanied by vomiting, and this may also occur in paroxysms without vertigo, often in association with headache.

Other paroxysmal disturbances are sometimes met with, especially of apoplectiform character, similar to those that occur in general paralysis of the insane. There may be attacks of simple coma, with pyrexia (even to 104°), or there may be unilateral convulsions, or the attack may be followed by transient hemiplegic weakness or rigidity. Occasionally the patient dies in such a coma, and, post mortem (as in general paralysis), no acute lesion can be found. Such paroxysmal disturbance, or some more trifling functional derangement, may precede the definite symptoms of the disease.

The general nutrition of the sufferer is often unimpaired, and sometimes there is an actual increase in the amount of subcutaneous fat. Trophic disturbances have been observed in rare cases,—local œdema, altered secretion of sweat, changes in the growth of hair or nails, chronic arthritis, and herpetic eruptions.

Course and Duration.—In some cases the course of the malady is uniformly progressive from the first to the last. More frequently periods of apparent arrest alternate with periods of progress, and its full development may be followed by arrest, actual or approximate, for years. The duration varies between two and fifteen years, the average being between three and six years. The most rapid case I have known was fatal a year and three quarters from the onset. The most common cause of early death is interference with the functions of the medulla oblongata, especially impairment of deglutition with its various consequences. In a few cases death occurs from failure of repiration. In the cases of longer duration the patients die from weakness, bedsores, kidney disease, &c., and in not a few instances death results, not from any effect of the disease, but from some intercurrent malady.

Pathology.—Compared with many of the other forms of sclerosis in the central nervous system, this variety is sharply distinguished by its irregular distribution. The islets are, as it were, scattered at random in seat, and they thus present a marked contrast to the "system-degenerations," in which a tract of sclerosis extends through the whole of a group of fibres having a certain function, and is sharply limited to these, or is restricted to a certain area of grey matter, or even to related groups of cells. The fact suggests that, whereas systemic sclerosis certainly commences by a wasting of the nerve-elements, to which the overgrowth of connective tissue is secondary (see vol. i, 2nd ed., p. 425), in disseminated sclerosis the primary change must be in the interstitial tissue, and the damage to the nerve-fibres must be secondary. The conclusion is supported by all we know of the pathology of the disease, by its aspect and its histological features, by the gradual way in which the white substance of the fibres wastes before the overgrowth of tissue about them, and by the absence of secondary degenerations unless the local process has a certain seat and a certain degree. The immediate local determining causes of the overgrowth cannot yet be even guessed at. It has been conjectured that it proceeds from the walls of the vessels, but whenever there is an overgrowth of interstitial tissue there is always a special increase adjacent to the vessels, and these may take a secondary share in the process even when there is no primary vascular change (see vol. i, p. 426).

The mystery of the origin of insular sclerosis, of the mechanism of its causation, and the nature of its pathological relations are increased by the fact that, different from system-degeneration as it is in the main, the two are not separated at all points. System-degenerations may be combined with insular sclerosis. The most common instance of this is the occurrence of primary atrophy of the optic nerves in disseminated sclerosis. This is a true system-degeneration of the nerves, similar to that which occurs in tabes. It is possible that in some cases there is also a system-sclerosis of the posterior columns of the cord in the lumbar region, so that tabes and insular sclerosis are combined, while some of the combined ocular palsies point to the same conclusion, and so also does the occurrence of the disease in families with a pure neurotic disposition, such as insanity indicates. Hence, different as the two processes are in their immediate pathology, they may possess some common relations in nature or causation, but our knowledge is still insufficient to enable us to make any definite statement on the subject. We may, however, bear in mind two very different sets of facts. One is the relation between the nutrition of the interstitial neuroglia and that of the nerve-fibres, especially of the medullary sheath, conspicuous in all processes of secondary degeneration, alike in general relations and in minute features. It is a converse relation, the decay of the one involving the growth of the other (see vol. i, 2nd ed., p. 396), but is not therefore to be passed as without significance. It seems alike influential whichever element in the process leads the way, as insular sclerosis illustrates. The primary atrophy of the optic fibres causes interstitial growth, and the scattered islets of the latter cause the nerve-fibres to waste.

A second fact which deserves to be borne in mind, although its significance is obscure, is that the only lesion which resembles insular sclerosis in general distribution, and sometimes in characters, is one that is apparently quite distant from it in etiology, the disseminated inflammation in the central nervous system produced by syphilis. This is always distinguished by its subacute onset, and usually by the caseation that it presents; but when such caseation is absent the aspect of the lesions is scarcely distinguishable from that of sclerosis. The illustration of the latter (Fig. 144) may be compared with the caseating foci shown in Fig. 125. I have seen a disseminated myelitis, without caseation, the aspect of which could scarcely have been distinguished from insular sclerosis; but it developed in a few weeks (immediately after treatment had removed a syphilitic tumour of the brain), and caused death in three months. These specific inflammations usually resist treatment, and are therefore probably due to a "toxine" produced by the organisms rather than to the latter—a fact which brings them into line with some systemic degenerations, such as tabes, although the latter begins in, and the former outside, the nerve-elements. We have here processes that bear a close analogy to those of insular sclerosis. But it is no more than an analogy, because the general history of this disease make it impossible for us to ascribe it to the acquired influence of either organisms or their products, unless in occasional and exceptional instances (as after an acute disease) which perhaps approach, in nature, such forms of inflammation as that met with in the subjects of syphilis. The common absence of distinct causation, the early adult age of the subjects, and the frequent neuropathic heredity, all point to some inherent condition, some disseminated congenital tendency of the nutrition of the neuroglia. The system-degenerations of the same period of life (such as that of "hereditary ataxy") we have seen (vol. i) must be ascribed to an inherent defect in the vital endurance of the tissues, and such degenerations may occur with insular sclerosis. Moreover, in connection with the apparent scattered tendency to an abnormal growth of the neuroglia we may remember, first, the facts already mentioned connecting the nutritional tendency of it and of the nerve-elements, which may involve similar liabilities to disease; secondly, the features of "gliomatosis" of the cord, and its active growth in early adult life; and thirdly, the modern theories (perhaps also facts) which ascribe tumours of many kinds to congenital tissue-elements, whose long quiescence may end without perceptible cause. In the present state of our knowledge it seems impossible to do more than offer such suggestions regarding the pathology of this disease. It may be noted that they include its chief, even different, features, its many varieties, and gradation into doubtful forms, and do not exclude the possible action, in rare cases, of acquired influences, such as those connected with acute specific maladies and general causes. Lastly, we can thus understand our inability to cure, and too often even to arrest the disease.

The chief general facts seem to be that insular sclerosis is a neuroglial disease, and that, apart from secondary degenerations, system-disease of the nerve-elements sometimes forms part of it. It is essentially a process of morbid growth, but may perhaps sometimes commence as an interstitial inflammation. Most morbid growths, however they may originate, when once set up, have a vital independence, and often a distinctly aggressive character, which carries them far beyond the range of their primary cause.

Diagnosis.—The great characteristic of the disease is the combination of the jerky inco-ordination and progressive weakness in the limbs with nystagmus. In such cases the diagnosis is easy. The inco-ordination differs from that of locomotor ataxy in the wildly irregular movements or tremors which, as it were, break out on any attempt at voluntary motion. The only disease in which this symptom exists in precisely the same form as in disseminated sclerosis is cerebral tumour (see p. 510). But these cases are distinguished by other indications of a tumour of the brain, and real difficulty in diagnosis can only arise from ignorance of the occurrence of the symptom in such cases.

The diagnosis from paralysis agitans depends chiefly on the symptoms which exist in that disease in addition to the tremor, on the regularity of the latter, even when considerable in range, and its locality, as will be specially described. The distinction does not depend on the relation of slight tremor to movement, or on the affection or freedom of the muscles of the neck. Hesitation between the two should be rare.

In some cases of general paralysis of the insane, the twitching contractions of the muscles occur in the limbs with unusual intensity, and may disorder voluntary movement in a manner very similar to that seen in disseminated sclerosis. The chief difference is that the spasm is most intense in the lips and tongue in general paralysis, and that the speech presents a drawling character, with hesitating phonation, while considerable mental change is almost invariable. A similar difficulty, at least in aspect, is presented by some cases of infantile brain disease, involving the motor cortex of both hemispheres. There may be jerky inco-ordination of the arms, identical in aspect with that of sclerosis, inco-ordination of the legs, and even a staccato articulation. Birth palsy (meningeal hæmorrhage) or thrombosis in the veins and (incomplete) in the longitudinal sinus, are the probable causes. The latter may have been the lesion in a case in which the symptom dated from a brain illness at fifteen months and ultimately resembled, very closely, those of insular sclerosis. But however close the resemblance, the history of such cases at once distinguishes them. The difficulty of the diagnosis may seem great at the first glance, but the onset, or development and careful observation of the several symptoms always shows the nature of the case.

The spastic paraplegia that results from insular sclerosis in the dorsal region of the spinal cord, and secondary degeneration below is not distinguishable from that met with in other chronic lesions of the same part, including primary lateral sclerosis. But characteristic symptoms elsewhere usually appear. The distinction of this disseminated dorsal myelitis, described in the section on Pathology, depends on the same considerations, but especially on the difference in the course of the two diseases, the subacute development of the paralysis in the disseminated inflammation.

Cases have been described by Westphal,* Leyden, and Langer,† in which symptoms closely resembling those of insular sclerosis have existed during life without any post-mortem lesion to which they could be ascribed. There is much doubt as to the exact nature of such cases, but they are too rare to be of much practical importance.

Perhaps the greatest real difficulty is presented by the cases in which there is only fine tremor in the hands with some weakness in the legs. The fact is certain that fine tremor may be combined with the other symptoms (see p. 548), and that which may co-exist with more typical disorder may also exist alone. A careful and repeated search should be made for any unequivocal symptoms, and these will often be found in the movements of the eyes. These cases are most common in girls, even when genuine, and they furnish the most frequent examples of the inevitable diagnostic problem, the distinction from hysteria. The difficulty is occasioned by the fact that an irregular tremor, occurring on movement only, is sometimes due to hysteria. While resembling that of sclerosis in irregularity, it is much smaller in range, and it is always accompanied by a tardiness of muscular effort, if the patient is desired to exert force, and there is the contraction of antagonistic muscles, described in the section on hysteria, which is of so much diagnostic importance. The variations in the symptoms, and the other indications described in the chapter on "Hysteria," seldom leave real room for a mistake. An error is especially easy when actual symptoms of hysteria preceded the onset of the disease, or occurred during its course. But hysterical tremor may generate a hopeless prognosis, and distress equally causeless and severe. The other error is the common one, and can only be avoided by the custom, which should be a habit, of ignoring any symptoms of hysteria until after a thorough search for those of organic nature, referred to above. Nystagmus may be safely accepted as evidence of more than hysteria, and this is one of the many maladies in which the symptom is of great diagnostic value.*

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* 'Archiv f. Psych.,' xiv, pp. 87 and 767.

† 'Wien. med. Presse,' 1884, p. 698.

PROGNOSIS.—The prognosis of insular sclerosis is exceedingly grave. It depends chiefly on the observed tendency of the disease. Yet the exceptions met with in most maladies are not unknown even in this disease, and should prevent a prognosis absolutely bad. One patient, who seemed unlikely to live many months, had improved at the end of a year and a half so as to be able to walk several miles. In cases in which there are periods of apparent arrest, the duration of life is likely to be longer than in those in which the progress of the symptoms is uniform. In the latter it is not probable that life will be prolonged for more than two years from the time at which the symptoms are sufficiently distinct to permit the diagnosis to be made with confidence. In the former the patient may live for several years, but in all cases much depends on the amount of care that can be given. The symptoms of greatest gravity are those of damage to the nerves of the medulla oblongata, especially difficulty in swallowing. The earlier such symptoms come on, the shorter is likely to be the duration of life.

TREATMENT.—Insular sclerosis is a malady for which, as its history shows, little can be done. The primary overgrowth of the interstitial tissue resists therapeutical influences more than does the primary degeneration of the nerve-elements. But it is probable that its causes vary, and, if so, some forms may prove more amenable than the majority. The treatment for the system-degenerations is that which most deserves a trial in the insular form, especially the nervine tonics, such as arsenic, nitrate of silver, quinine, &c. The hypodermic injection of arsenic has been recommended by Eulenburg, but the evidence that this method of administration has a special influence is insufficient. Occasionally these drugs seem to retard the progress of the disease, but an arrest of its progress is seldom lasting. Solanine, in doses of $\frac{3}{8}$ gr., has been recommended by Grasset as lessening the tremor. Hydropathic treatment, and the use of electricity in various forms, have been advocated in Germany, but their influence is exceedingly doubtful. In the case of a peripheral palsy, such as that of the face, due to sclerosis of the nerve, the question presents itself whether the muscles should be subjected to electrical treatment. As a rule it is not worth while to give the patient the pain and inconvenience of the applications. The disease is so often progressive, that no recovery of the nerve can be anticipated; hence electrical treatment, however prolonged, can have no influence in modifying the condition, and there is not the same reason for applying it to the muscles that exists in the case of lesions from which the nerve-fibres may recover. It is of course desirable to maintain the patient's general health in as good condition as possible, and to avoid all depressing influences. In the case of women, pregnancy is especially undesirable; the malady is apt to make more rapid progress during the later period of pregnancy, and sometimes after delivery. When more is known of the causes and essential pathology of the disease in different cases, more rational methods may brighten the therapeutic prospect.

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* Nystagmus has been said to occur in hysteria, but the assertion is of very doubtful truth. In an extensive investigation into the distinction between these two diseases, Sougues could not discern it in any case of hysteria ('Nouv. Icon. Salpêtrière,' 1892).

DIFFUSE SCLEROSIS.

If the connective-tissue elements are increased through a considerable area in any part of the brain, the sclerosis is said to be "diffuse," in distinction from the small islets of the disseminated or insular sclerosis just described. Diffuse induration may involve either the white substance or the cortex, part or the whole of one hemisphere or of both, the cerebrum or the cerebellum. Sclerosis may also be secondary to some other lesion, such as softening, the evidence of which (as a cavity adjacent) is still conspicuous; but such forms are not here considered. Two varieties must, however, be distinguished as fundamentally distinct,—those in which there is, and those in which there is not, diminished bulk of the affected part. In the latter, as a rule, the change may involve the white substance only or the grey matter, or both, and the induration is primary; in the former the cortex is chiefly affected, sometimes with a secondary implication of the deeper structures. In these cases the diminution in size constitutes what is called "atrophy of the brain," general or partial, and it is often very difficult to say in which class the lesion should be placed, and by which term it should be named, "sclerosis" or "atrophy." But it is certain that, in most cases of partial disease, and in many in which the affection is general, unilateral or bilateral, the influence causing atrophy is primary and the induration is secondary, the result of the wasting of the nerve-elements which entails the overgrowth of the interstitial tissue. Such sclerosis is very common as a secondary change. Sclerotic induration, for instance, follows the damage to the cortex produced by the meningeal hæmorrhage that occurs during birth (see p. 413). It is a necessary consequence of interstitial inflammation, however produced, and so is found in the cortex, when this is diseased in cases of acquired infantile hemiplegia, whether the primary process is venous thrombosis or acute inflammation. If any influence causes a general arrest of the growth and development of the cortex of the brain, it induces this combination of atrophy and sclerosis, the cortex being the part on which such influences seem chiefly to act. This state may result from a concussion of the brain in childhood, from an actual focal lesion, or from the effect of severe and repeated epileptic fits. But in all such conditions we must regard the sclerosis as secondary and not primary, the first effect being produced on the nerve-elements; and the failure of their nutrition or development entails an overgrowth of the neuroglia, which ultimately becomes firmer than in some other conditions.

In the cases that may be regarded as primary sclerosis of the brain there is little or no diminution in the size of the part, and no indication of an affection of the nerve-elements to which the increase of connective tissue is secondary. They differ, however, so much in their features, causation, and symptoms, that it is difficult to classify them into definite forms until many more carefully observed facts have been collected. In one group of cases, met with almost exclusively in syphilitic children, there is induration of the grey cortex, usually through considerable areas, sometimes confined to a single convolution, rarely in small nodules.* The tint of the grey matter is but little altered, but the change in its consistence is very distinct to the touch. When this affection is of long duration there may be a trifling diminution in the bulk of the part. There may be slight thickening of the superjacent pia mater, but there is no arterial disease distinctly related to the affected region. Very rarely the central grey matter has been the seat of such diffuse sclerosis. The microscope shows an increase in the neuroglia, and wasting of the nerve-cells. A similar change is seen in the sclerosis that attends cortical atrophy. The interstitial tissue has been found to present the aspect of a finely granular network, and the axis-cylinders of the included nerve-fibres have had a peculiar irregular outline.†

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* It is well described by Bury and Barlow, Tuke's 'Dict. Psych. Med.,' ii, 1261.

† Schmaus, 'Virchow's Archiv,' Bd. cxiii.

The white substance is not often the seat of sclerosis in congenital syphilis, except in very severe and extensive cases; and cortical sclerosis seems to be equally rare in the acquired disease of adults. But I have seen, in this affection, a diffuse induration of extensive tracts of the centrum ovale of each hemisphere, causing increased consistence, readily felt, but with little change in aspect. A diffuse, almost universal sclerosis has been observed in a subject of chronic alcoholism, associated with induration of the kidneys and other parts.* Lastly, areas of diffuse sclerosis have been found, in rare cases, between and connecting the islets of the disseminated form,† a condition that is perfectly consistent with the pathological suggestions made on a preceding page regarding this variety.

The symptoms of diffuse sclerosis have varied much in different cases, so much indeed that its existence can only be guessed at—except, perhaps, in some cases of congenital syphilis. In all cases of cortical sclerosis in childhood, mental defect exists in proportion to the extent and, when limited, to the seat, of the lesion. Every grade of defect, down to profound idiocy, is observed. When the central region is affected, motor symptoms become conspicuous, loss of power on one side or on both, convulsions, and especially contractures of the limbs and spontaneous movements. In adults, the symptoms have been, on the whole, similar, but have presented great variations. Hemiplegia, contracture, attacks of clonic spasm or tremor, headache, giddiness, vomiting, paroxysmal loss of consciousness, and slowness of speech, have been met with, some frequently. Among the latter, the attacks of spontaneous movement have been thought to be the most characteristic (Strümpell).

Much more must be ascertained regarding the affection before any prognostic data can be obtained, or rules for treatment laid down, beyond those indicated by its apparent causes, and such as are suited for any chronic cerebral affection.

MILIARY SCLEROSIS.

The term "miliary sclerosis" was first applied by Drs. Batty Tuke and Rutherford to a lesion found by them in the white substance of an atrophied cerebellar hemisphere in an insane patient. The lesion was chiefly microscopic, and was only discovered after the brain had been hardened. It consisted of minute spots of degeneration, in which there was little evidence of a process deserving to be called "sclerosis." The term has been since used by others to designate another microscopic lesion, which was at first thought to be really pathological, but has since been abundantly proved to be of artificial origin—an

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* Strümpell, 'Arch. f. Psych.,' 1879.

† See Westphal, 'Arch. f. Psych.,' viii; and Kelp, 'Deut. Arch. f. kl. Med.,' x, 1872. Cases have also been recorded by Schule, Jolly, and F. Schulze.

"artefact," as such changes have been termed. The most common appearance is that in minute areas the tissue remains unstained by reagents, and presents a striking contrast to the well-stained vicinity. On close inspection, however, the tissue-elements can be traced in the unstained area; and it is evident that the difference between these spots and the adjacent tissue is due to the absence of staining. This appearance is chiefly seen when spirit has been employed in hardening. The central organs ought never to be placed in spirit, at any rate, after the first twenty-four hours. This is only one of many artificial changes produced by spirit, which are often very puzzling to the observer.

It is questionable whether the term "miliary" ought to be applied to a purely microscopic lesion; it is more accurately used to designate changes resembling in size "miliary" tubercle. But that there is a form of sclerosis of the brain which is truly "miliary" is evident from one well-marked example that has come under my notice. In this case, throughout both hemispheres of the brain, and in all parts of them, the cortex contained minute reddish-grey spots at the junction of the grey and white substance. In tint they were a little darker than the darkest grey substance, and their aspect was exactly like that of the morbid tissue in insular sclerosis, and equally distinct in the recent state. In size they varied from that of a mustard seed, or a little larger, to the smallest point visible; large areas, one eighth of an inch in length, were formed here and there by the coalescence of the smaller spots. Their size and distribution are shown in Fig. 145, A. None could be seen in the white substance of the hemisphere, but they were found in the central ganglia, especially in the outer part of the lenticular nucleus (B) and in the claustrum (C). None were found elsewhere in the brain, except two beneath the corpora quadrigemina. Under the microscope the centre of the large areas consisted of a delicate fibrous tissue with abundant spider-cells and small round nucleated cells. In the fibrous tissue were small cavities, especially at the periphery of the larger spots and throughout the smaller ones, so that the tissue had a somewhat sponge-like aspect. No connection could be traced between most of these areas and vessels, but in places a similar change, of spongy aspect, could be seen along the side of a vessel, sometimes extending for a considerable distance. Thus there was a wasting of the nerve-elements, and an increase in the connective tissue in the affected areas.

I have not met with the record of any quite similar case, but an analogous lesion has been observed in a few cases of general paralysis of the insane, invisible, however, in the recent condition.*

In my case there was no mental derangement. The patient was a man aged fifty, and the first symptom was general weakness of the limbs, accompanied with some rigidity and a few left-sided convulsive attacks, beginning in the muscles of the shoulder. Then the speech became "mumbling" and unintelligible; coma set in, and after a few days of unconsciousness he died. He had also some cardiac weakness, enlargement of the liver and spleen, and general anasarca without albuminuria. The duration of the nervous symptoms was about ten weeks. No cause for the disease could be traced, but he had had constitutional syphilis many years before.*

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* See Grieff, 'Archiv f. Psych.,' xiv, p. 287; and Simon, ib., Bd. ii.

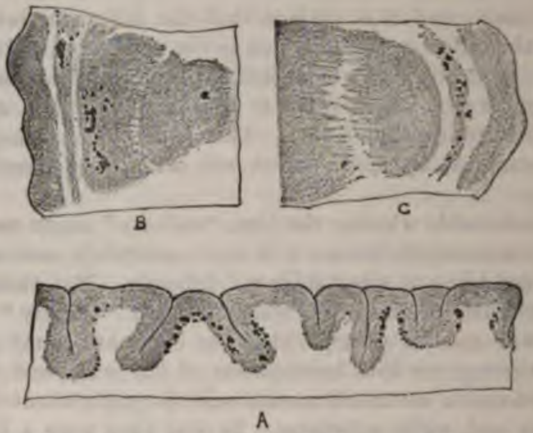

FIG. 145.—Miliary sclerosis of the brain. Sections, A, of the cortex, B, of the left, and C, of the right lenticular nucleus and claustrum. The black dots represent the size and distribution of the minute foci of sclerosis.

ASSOCIATED PALSY OF THE BULBAR NERVES.

It has been mentioned that, among the cranial nerves, we may recognise two special groups, subserving associated functions and liable to associated disease. Each group is motor: one comprehends the nerves of the eyeball muscles, the associated palsy of which has been described at p. 194; the other includes the nerves to the complex series of muscles of the upper part and orifice of the respiratory and alimentary passages—the mouth, fauces, pharynx, and larynx. These arise from tracts of grey matter in the lowest part of the "middle brain" and the junction of this with the spinal cord, the "medulla oblongata," or "bulb." The associated palsy of these "bulbar" nerves has now to be considered. The most common form is due to a degenerative process similar to that which occurs in the spinal cord. But, as we have already seen, a similar paralysis may result from a sudden lesion in the same part—sudden or "apoplectiform" bulbar paralysis. It is sometimes termed the "acute" form, but there is also a rare variety due to acute inflammation of these structures, for which the epithet "acute" is needed. On account of the general resemblance of the symptoms it will be convenient to mention the symptoms of the acute and sudden forms in connection with the common, chronic variety. As we shall see, disease of the nerves may cause a like effect, and so also (although by a very different mechanism) may disease of the two cerebral hemispheres. These forms (especially the latter), from their superficial likeness to bulbar disease, but essential difference from it, have been called "pseudo-bulbar paralysis."

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* Fuller details of this case will be found in the 'Lancet,' 1886, January 23rd, p. 145.

The nerves, in the distribution of which the symptoms occur, are the fibres of the facial for the lower part of the face, especially for the orbicularis oris, the hypoglossal for the tongue, the spinal accessory for the larynx, and probably also for the palate, and the glosso-pharyngeal or vagus for the pharynx. It deserves note that the facial nerve occupies, as it were, an intermediate position between the two nerve-groups above mentioned as liable to associated disease; it supplies, on the one hand, the muscle that closes the eyelids, and on the other, that which closes the mouth. But the orbicularis palpebrarum does not seem to be very closely associated with the eyeball muscles, for it does not suffer with them in disease of their nuclei. Its functional association with the eyeball muscles is unimportant; it is only a small part of these relations, and does not apparently include its action in protecting the eyeball. But the orbicularis oris and the transverse muscles of the front of the tongue have a functional association closer, perhaps, than any other two muscles in the body equally distinct. Neither can be put in action without the other (see p. 50, note). The orbicularis is always involved in degenerative disease of the hypoglossal nucleus, and escapes in disease of the chief nucleus of the facial, as in a case of acute atrophic ("infantile") paralysis in which the face was involved (see pp. 231 and 240).

The central connection of the chief parts that are affected in the disease is well illustrated by the combination of paralysis of the tongue, palate, and vocal cord that results from disease of the nerve-roots on one side of the medulla (p. 307). In central disease of the medulla the paralysis has the same distribution as in a lesion of the nerve-roots, but it is almost always bilateral, and, in addition, the pharynx is often, and the lips are always, paralysed. These escape in disease of the roots, because the labial nerve-fibres, although they seem to arise from cells in the medulla, ascend to the lower part of the pons, which they leave by the facial trunk. The most characteristic and complete distribution is seen in the cases of chronic degeneration in which the nerve-cells suffer according to their functional nature. The chief centres concerned lie, however, so near together in the medulla, that random processes, such as softening or hæmorrhage, may damage them on both sides, and cause palsy of the same distribution as that which results from primary degeneration. Hence all forms are included under the term "bulbar paralysis," but only the chronic cases and the inflammatory form can be regarded as constituting distinct maladies.

CHRONIC BULBAR PARALYSIS.

The chronic bulbar paralysis, the "labio-glosso-pharyngeal" (or "laryngeal") paralysis, was first described in 1859 by Du Mesnil, and systematically by Duchenne in 1860, while this, like so many of Duchenne's investigations, became widely known chiefly through the lectures of Trousseau. The awkwardness of the descriptive name given by him, led Wachsmuth, in 1864, to propose to call the disease "progressive bulbar paralysis," a designation that has come into general use. The dependence of the symptoms on degeneration of the nuclei of the medulla was established especially by the labours of Charcot's pupils and of Leyden.

CAUSES.—The disease occurs chiefly in the second half of life, although it is sometimes met with in middle life, and in a few anomalous instances has developed in childhood, at seven, eight, and ten, and occasionally in several children of the same family.[*] But most of the sufferers have been between fifty and seventy when the disease commenced. Males are rather more liable than females. Direct inheritance is practically limited to rare cases in which the affection forms part of progressive muscular atrophy,[†] but indirect inheritance, shown by other related neuroses, may often be traced. Moreover, a strange family disposition has been observed in cases occurring in early life. It does not seem to be a consequence of syphilis. Immediate causes can be traced in only a minority of the cases; the most frequent are mental depression and anxiety (sometimes combined with physical exhaustion, as in nursing a relation through a grave and fatal illness), exposure to cold, debilitating influences, such as insufficient food, and injury, such as a blow upon the back of the neck. Over-use of the muscles has been supposed to be a rare cause; the symptoms developed rapidly in a man after playing on the clarionet for two whole nights, but the patient at the same time had been exposed to cold (Stein). In a considerable proportion of the cases, no cause, immediate or remote, can be traced. It is not unlikely that many of these will ultimately be found to depend on some toxæmic influence. I have known the characteristic form to follow lead-poisoning in advanced life, and also partial bulbar paralysis to be associated with partial nuclear ophthalmoplegia in early adult life as a late sequel to diphtheria.*

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[*] At seven and eight, two sisters and a brother, the offspring of intemperate cousins, Moty, 'Gaz. des Hôp.,' 1889; at eight, Remak, 'Berl. Ges. de Psych.,' 1890; at eleven, Hoffmann, 'Cent. f. Nervenkr.,' 1889; at twelve, Blumenthal, 'Inaug. Diss.,' Dorpat, 1884. Many supposed cases in early or middle life have been due to tumours.

[†] As in three cases in one family described by Bernhardt, an instance observed by Berger, and one in old age (mother and daughter) by myself.

The relation to progressive muscular atrophy has been already mentioned. The bulbar disease is indeed part of the spinal affection, the grey matter of the medulla undergoing the same degenerative change as that of the spinal cord. Lastly, a limited acute nuclear inflammation may be followed by slow degenerative extension.†

SYMPTOMS.—The distribution of the symptoms, indicated by the name given to the disease by Duchenne, has been already mentioned. The lips, tongue, throat, and often the larynx, are paralysed on both sides. The symptoms are, so to speak, grouped about the tongue as a centre, and it is in this organ that the earliest weakness is usually manifested. In a few cases the distinct paralytic symptoms have been preceded by some discomfort or pain at the back of the head. The onset of definite symptoms is gradual; the cases in which there is a sudden or even acute onset are not examples of this form. The first symptom is generally a trifling indistinctness of speech, due to imperfect articulation of those sounds in which the tongue is chiefly concerned, the lingual consonants *l*, *r*, *n*, and *t*, and afterwards *s*. The commencing change in articulation is noticed only when the parts are fatigued by use, and the patient may be conscious that fatigue is produced with undue readiness. The early difficulty in articulation is often ascribed to some dental imperfection, or to dentists' procedures undertaken to lessen the imperfection. The tongue at first can still be protruded, although sometimes not so far as normal, and there may be a difficulty in bringing its tip firmly against the roof of the mouth for the articulation of the linguo-palatine consonants, as *t* and *d*, while the continuous sibilant *s* can be perfectly articulated. The lips become weak with or soon after the tongue, and some difficulty in swallowing is often added; the latter may, indeed, be the earliest symptom, or it may only come on after articulation has become much impaired. In consequence of the weakness of the lips the power of whistling is lost, and there develops a difficulty in the pronunciation of the sounds in which the lips are chiefly concerned, *o*, *u*, *p*, *b*, and *m*. The lips are not brought together so perfectly, or separated so promptly, as is necessary for the pronunciation of the labial explosives, and hence *b* and *p* become *m* and *v*. The power habitually exerted is generally less than the patient can exert if he tries. A word can often be articulated perfectly by a deliberate effort when the habitual articulation is very imperfect. The difficulty in speech is soon increased by the weakness of the palate, which ceases to shut off the nasal cavity. Swallowing gradually becomes difficult; the weakening of the tongue impairs the first part of the act of deglutition, that in which the food is rolled back into the pharynx by the application of the tongue to the roof of the mouth; the paralysis of the palate permits the regurgitation of liquids into the nose, and the weakening of the pharyngeal muscles further interferes with the movement of the food.

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* In a case described by Stadthagen, diphtheria at four was followed by the common paralysis of the palate, and this by wider symptoms of bulbar palsy. When the patient came under observation, some years later, there still was paralysis of the lips and palate, accompanied by weakness and contracture in the limbs ('Archiv f. Kinderheilk.,' Bd. v).

† Reinholdt, 'Arch. f. klin. Med.,' 1889.

As the paralysis increases, closure of the mouth becomes impossible, the lower lip droops, and the muscles of the chin often become feeble. The elevator of the upper lip and zygomatics either escape, or are so much less affected than their labial opponents, that the zygomatic muscles undergo contracture, and a very pronounced naso-labial furrow becomes a characteristic feature of the disease. Saliva dribbles out of the open mouth, and the patient has to keep a handkerchief constantly in his hand.* The tongue can no longer be protruded; only the tip can be projected over the lower teeth. Mastication is difficult, because the tongue can no longer guide and keep the food between the teeth. The palate may hang motionless, or it may still be raised a little by the will. Articulate speech at last becomes impossible, and the only expression remaining to the patient is laryngeal phonation, slightly modulated, and broken into the rhythm of formless syllables, the meaning of which can be hardly more than guessed at. Swallowing becomes more and more difficult. Semi-solids are usually swallowed better than liquids, because there is less risk of their escape into the nose or larynx, and better than solids, particles of which are more apt to enter the larynx from the increasing weakness of the epiglottidean muscles, which fail to reflect the epiglottis during the act of deglutition. The muscles of the vocal cords generally suffer now, if not before; the voice is low-pitched from the defective approximation of the cords, and the cough is imperfect from the feebleness of closure. At first the glottis may be closed only in one of several attempts to cough. The laryngeal palsy rarely becomes complete, and it is still rarer for the power of abduction to be specially lost, common as abductor palsy is in some other forms of central degeneration. I have, however, seen abduction completely lost on one side when it was preserved on the other. Thus in some cases the pharynx suffers, in others the larynx, but both are usually involved in the later stage. The difference seems to correspond to a difference in the localisation of the affection, according to the functional relation of the parts; in some cases those for articulation and in others those for deglutition being apparently primarily involved. Since all the muscles concerned and the structures related to them are in great part the same in the two sets of cases, there is considerable general correspondence in the symptoms and lesion.

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* It is often thought that there is an increase in the secretion of saliva, and "salivation" is said to exist, but it is doubtful whether the amount is larger than the normal. A pathological increase has been asserted very strongly by some authors; Schulz supposed that, in one case under his care, the secretion was increased to six times the normal quantity.

There is no loss of sensibility in any of the affected parts, and taste is unimpaired, but the reflex action in the throat, so active in health, ultimately fails from the motor changes. The palate or fauces, and even the larynx, can be touched or tickled without exciting the spasm that is normally produced. Sometimes this loss of reflex action precedes the other symptoms of the disease (Krishaber). It increases very much the risk of the entrance of food into the larynx. But reflex action is not invariably lost; Erb twice found it preserved in the palate and pharynx to a late stage of the disease.

Alterations of nutrition are chiefly visible in the tongue, and its condition varies much in different cases. In some the organ is large and broad throughout, although soft and flabby to the touch. In other cases it is conspicuously wasted, and the surface is deeply furrowed and wrinkled from the loss of the muscular substance (Fig. 146). In some cases the lips retain their normal size; in others they are conspicuously thin. Electric irritability is seldom much changed; the muscles, even when greatly wasted, may still react to faradism with readiness. Rarely, in rapid cases, there is loss of faradic and preservation of voltaic irritability, and Erb has observed the increased and altered reaction to voltaism, when the faradic excitability is normal, of the mixed (or "middle") form of reaction (see vol. i, p. 26). Symptoms of motor irritation, such as convulsion or spasm, are absent, but choreiform twitchings may occur on an attempt to speak, chiefly due apparently to the influence of emotion.

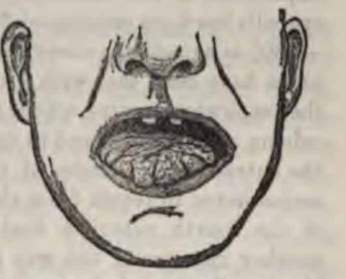
FIG. 146.—Wasting of the tongue, and maximum protrusion, in a case of chronic bulbar paralysis.

Thus there appear to be two forms, one with wasting and loss of the reflex action, the other with no wasting and sometimes with distinct evidence of excess of muscle reflex action, at least so far as the muscles of mastication are concerned. The occurrence of the latter form cannot be doubted even when allowance is made for the concealment of atrophy by fat. It is apparently analogous to the spastic paralysis met with in the limbs. Not only may the "jaw-jerk" be increased in this form, but I have once seen a distinct clonus in the muscles of mastication, obtained by depressing the jaw.

The intellectual faculties, as a rule, are unimpaired. The only alteration that is common is a curious emotional mobility; laughter or tears are evoked by trifling causes, and the immobile face and unmodulated tone may render the laughter strangely grotesque. But the unhappy sufferer is painfully conscious of his state; and his distress is increased by the inability to express it, except by the laryngeal gestures to which speech is reduced. His consciousness of inability often renders him indisposed to make even an attempt to express himself.

Progressive bulbar paralysis, as already mentioned, is often associated with progressive muscular atrophy in the limbs and trunk, accompanied, it may be, with the symptoms of lateral sclerosis of the cord. Either the bulbar or spinal symptoms may lead the way, and dominate the aspect of the case. Towards the close of a general progressive atrophy the mouth and throat muscles may suffer, or when these are first and considerably affected, wasting may show itself in the muscles of the limbs. Less commonly, bulbar paralysis is associated with spastic paralysis of the limbs and slight wasting; lateral sclerosis is then found after death, with only slight changes in the anterior horns.

Even in cases not accompanied by general atrophy, the paralysis may spread beyond the muscles of the lips, tongue, and throat. The rare implication of the muscles of the upper part of the face and of the eyeballs has been mentioned.* Excessive frequency of the pulse (150—160) is sometimes observed towards the close, and attacks of dyspnœa have been met with from the extension of the degeneration to the respiratory centre, which also, towards the close, seems to produce œdema of the lungs, and to facilitate the low pneumonia produced by the entrance of particles of food. It is noteworthy that glycosuria seems never to result from the degenerative changes beneath the floor of the fourth ventricle that constitute this disease. This may be another instance of the way in which functional relation determines the distribution of the morbid process. Occasionally the muscles of mastication become weak, and the pterygoids, which move the jaw laterally, are said to suffer first, and often only (Duchenne). Optic nerve atrophy has been noted in very rare cases.†

Among the indirect effects of the disease are the weakness and emaciation that result from the imperfect supply of nourishment consequent on the difficulty of swallowing, and the bronchitis that is apt to be set up by the passage of particles of food into the air-passages.

The *Course* of the disease is progressive, although not uniformly so. From time to time its progress is retarded, and it may seem for weeks, or even for months (seldom for a year or so), to be at a standstill. The hopes thus excited are usually baseless, for the disease again increases, often at a more rapid rate. An intercurrent malady, which lessens the patient's strength, accelerates the progress of the disease. But the cases which occur in the first half of life occasionally become arrested before reaching an extreme and dangerous degree, and the duration of this later stage may be indefinite.

The *Causes of Death* are gradual weakness from inanition; broncho-pneumonia, from the entrance of food into the air-passages; failure of respiratory power from associated atrophy of the muscles of the trunk, or the effects on lungs and heart from the extension of the degeneration to the pneumogastric centres.

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* Attention has lately been especially directed to it by Guinon and Parmentier, Nouv. Icon. de la Salpêt.,' 1890 and 1891.

† Ritchie, 'Glasgow Med. Journ.,' 1888; Robin; Galezowski (quoted by Ritchie).

Pathology.—In the affected muscles there are changes similar to those seen in progressive muscular atrophy of the limbs. The fibres may present extensive granular and fatty degeneration, or may be simply narrowed. Usually some fibres are much affected, and others but little, so that empty sarcolemma sheaths and normally striated fibres may lie side by side. There is an increase of the nuclei of the sheaths and of the interstitial tissue, and the latter may present an abnormal accumulation of fat. Masses of reddish pigment, the product of the degeneration of the fibres, lie between them. The nerve-endings in the muscle are also degenerated. The motor nerve-trunks are grey and soft, and the microscope shows degeneration of the nerve-fibres and increase of the interstitial tissue.

The medulla oblongata itself generally appears normal to the naked eye; rarely there is slight diminution in bulk. The important morbid appearances are revealed only by microscopical examination. There may be distinct atrophy of the fibres of the hypoglossal and other nerves within the medulla. In the motor nuclei, changes are found similar to those presented by the grey matter of the cord in progressive muscular atrophy. There is wasting of the nerve-cells, many of which lose their processes and shrink. The interstitial tissue is altered in various degree, and contains granule-corpuscles and other products of the degeneration of the nerve-elements in early cases; in some instances there is a conspicuous increase in the connective-tissue elements; the vessels may be increased in number and their walls thickened. But cases vary remarkably in this respect: in some, only a wasting of the nerve-cells can be discerned; while in others the aspect is that of an actual interstitial inflammation, even involving minute extravasations in the early stage,* and yet limited to the motor nuclei. The variations suggest that there are parenchymatous and interstitial forms.† This difference may explain some facts of limitation or irregularity of the symptoms.

Degeneration is often found in the anterior pyramids, just as the pyramidal tracts are commonly degenerated in the corresponding affection of the anterior cornua of the spinal cord, which so often co-exists with the bulbar degeneration. In such combined cases the sclerosis of the pyramidal fibres may be traced through the pons and cerebral peduncles. When, as is commonly the case, there is muscular atrophy elsewhere, the spinal cord presents corresponding alterations in the grey and white substance. It is probable that, in some cases of bulbar paralysis, the disease is confined to the pyramidal fibres, as in cases of lateral sclerosis of the spinal cord causing spastic paralysis.

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* Eisenlohr, 'Arch. f. Psych.,' 1879, p. 34.

† Compare "Acute Polio-myelitis," vol. i.

The degeneration in the bulbar nuclei is constant and most considerable in the hypoglossal nucleus, next in the adjacent part of the nucleus of the spinal accessory nerve, and it is usually found in less degree in the nuclei of the vagus, seldom in the nucleus ambiguus or glosso-pharyngeal, or in the motor nucleus of the fifth nerve, or in the chief facial nucleus (fig. 36, p. 48). The collections of small cells in front of and behind the hypoglossal nucleus* usually escape. Degeneration of some of the fibres of the raphe has been occasionally noted at the level of the hypoglossal nucleus. Considerable degeneration has been found in the fibres of the "loop" of the facial nerve, even when the cells of the chief facial nucleus were but little affected —a mysterious fact. The posterior longitudinal fibres may be normal or partly degenerated† (in proportion, it is said, to the affection of the hypoglossal nucleus); especially the longitudinal fibres suffer in the inner part of the reticular formation which are supposed to continue the anterior ground fibres of the cord. The degeneration is always bilateral.

The grouping of the symptoms according to function makes it practically certain that the incidence of the lesion is determined by function, that it begins, as a rule, in the nerve-elements, and follows their functional relations in its extension. The exact degeneration on which the paralysis of the orbicularis depends is the most obscure point. Lockhart Clarke believed that the small cells above the hypoglossal were connected with the facial nerve, and that it was their disease that causes the paralysis of the lips. Later researches have failed to confirm this, but it is highly probable that the orbicular fibres do descend to the neighbourhood of the hypoglossal nucleus, and may even arise from its cells (see p. 50); although it is impossible to trace them among the many horizontal fibres of the reticular formation, in which they probably pass downwards, perhaps reaching it by the raphe; into this many fibres of the facial nerve may readily be traced at the upper part of the "loop."‡ The fibres of the anterior pyramids and pyramidal tracts in the pons and crura have been found degenerated, chiefly in the cases in which there has also been atrophy or palsy of the limbs; the degeneration of the pyramids is then the rule (see vol. i, p. 490).

The probability has been already mentioned that, as Duchenne originally maintained, there are two varieties, the atrophic and the paralytic; and that, as already mentioned, in some of the cases without obvious wasting, the primary disease is not in the motor cells, but in the fibres that connect these with the cerebral hemispheres, or in

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* The former is often termed "Roller's nucleus."

† See Muratoff, 'Cent. f. Nervenk.,' 1891, p. 514.

‡ The tongue and lips have been paralysed together and alone on one side, with the reaction of degeneration (Grugia and Mattencei, 'Arch. Ital. Mal. Nerv., 1887).

the lower extremities of these fibres within the nuclei. We have seen that the pyramidal fibres may be degenerated, and these fibres are homologous with those that constitute the upward path from the nuclei to the cortex. In cases in which these fibres, and not the nuclei, are diseased, there should be no loss of reflex action, and we have seen that, in some cases, the reflex action is preserved. Moreover, in some of these cases there is marked excess of myotatic irritability in the muscles of mastication, even, as we have also seen, so great as to permit a jaw-clonus to be obtained—distinct evidence of the degeneration of the fibres of the upper segment of the motor tract for these muscles (see vol. i, p. 201). The other parts specially paralysed do not permit this muscle reflex action to be tested. Thus these cases will constitute the bulbar homologue of primary lateral sclerosis of the cord.

Atrophic bulbar paralysis must be regarded as an affection practically identical with the progressive muscular atrophy that is so often associated with it. The difference between them depends only on the seat of the morbid process, and not at all on its nature.* The disease is a degeneration of the lower segment of the motor path for the muscles affected, and in some cases it may be a degeneration of the whole path, upper and lower segments, or of the upper segment alone, when there is no wasting. Although an affection of the cortical cells has not yet been found, it is highly probable, from the analogy of progressive muscular atrophy. In other cases, again, there may be a combination of the two conditions analogous to those in which atonic atrophy in the hands is associated with tonic palsy in the upper arms (see vol. i, p. 480).

The relation of the degenerative changes to the function of the affected cells, so conspicuous in the distribution of the symptoms, merely, of course, means that a certain common function is associated with a common special susceptibility, on the part of the cells, to suffer from a defect in nutrition, which is progressive in character, and tends to increase to destructive degeneration. It is essentially a defective vitality, and, as such, is associated with an exuberance of growth in the interstitial elements, analogous to that which is seen in the elements surrounding a degenerating nerve-fibre. The real pathology of the disease is the cause which induces the nutritional changes, and of that we know almost nothing. The possible influence of toxic conditions should always be remembered.

DIAGNOSIS.—The distribution of the palsy, its bilateral character, its gradual onset and progressive course, separate the disease with sufficient sharpness from most other maladies. The slow onset distinguishes it from acute lesions of the medulla, which may cause the symptoms of similar character and distribution described in the next section. The chief difficulty is presented by organic diseases of the medulla, which may also cause "bulbar palsy" of slow onset. The most frequent of these is a tumour in or outside the medulla, or a narrowing of the foramen magnum by thickening of the bone or enlargement of the odontoid process, damaging the bulb by invasion or compression, or compressing the nerve-roots; but the symptoms thus produced seldom present the perfect bilateral symmetry that characterises the degenerative affection. One side is affected first or most, the difficulty of swallowing preponderates over the other symptoms, and the lips usually escape altogether. Moreover, headache is usual, and convulsions are occasionally met with.

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* The essential identity of the two diseases was first urged by Kussmaul (Volkmann's 'Clin. Lect.,' No. 54, 1873).

Tumours within the medulla sometimes give rise to greater difficulty, but the cases in which a growth acts on both sides in such an equal manner as to cause perfectly symmetrical symptoms are excessively rare, and there are usually other indications to help the diagnosis,—either there are other indications of a tumour, or the patient is so young as to render the degenerative disease highly improbable. Insular sclerosis, involving the medulla, is seldom so symmetrical as to give rise to real difficulty, and there are always indications, in other parts, of the morbid process.

Chronic lesions in both cerebral hemispheres may cause symptoms resembling those of bulbar palsy, constituting a chronic form of "pseudo-bulbar paralysis." Although such an effect is far more rarely produced by chronic than by acute lesions, it has been caused by symmetrical sclerosis of the two hemispheres; * the chief distinctions are afforded by the development of symptoms first on one side and then on the other—the affection of the limbs, as well as the face, in distinct double hemiplegia,—and by other symptoms indicative of the nature of the morbid process. Reflex action is preserved in the parts paralysed, and these are never wasted, but alone these points are not sufficient for the diagnosis, although they may be allowed weight in support of other indications.

Prognosis.—In every case of gradual onset the prognosis is most grave. The affection is so progressive in its tendency, and the parts affected are so important for life, that the disease almost invariably leads to death. This is especially true of the senile form, and of that which supervenes on spinal muscular atrophy. When the affection begins in middle life, arrest may be regarded as a possible event, and it is not improbable in the rare cases that come on in late childhood, or follow some disease that has nerve-degeneration for its occasional sequel. The possible cause, as well as the obvious course, must, therefore, be considered in the prognosis.

Treatment.—Although experience shows that we can scarcely expect, in any case, that our treatment will have an appreciable influ-

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* Jolly, 'Arch. f. Psych.,' iii, 1872.

ence on the disease, we may strive at least to retard its progress, and employ such measures as, from their action on the nervous system, are most likely to produce this effect. It is probable that cases will occasionally be met with in which the morbid tendency is less strong than it usually is, and in which treatment may have some influence. Nervine tonics, quinine, strychnine, arsenic, phosphorus, or nitrate of silver may be given, or hypodermic injections of strychnia ($\frac{1}{60}$ gr.) may be administered, as recommended for progressive muscular atrophy (vol. i, p. 497). The latter is especially indicated in the non-senile forms. In advanced cases I have known transient improvement to follow the injection of strychnia combined with minute stimulant doses of morphia ($\frac{1}{36}$—$\frac{1}{24}$ gr.). The power of swallowing has been increased by this treatment, but unfortunately the effect has not been permanent. To lessen the flow of saliva, belladonna or atropine has been given, but seldom with effect.

Electricity has been extensively used. With the view of influencing the morbid process in the medulla, the voltaic current has been passed from one mastoid process to the other. In order to maintain the nutrition of the muscles, either faradism or voltaism may be employed, for they respond to either in the majority of cases. It may be applied to the tongue, lips, or pharynx; to the last by placing the positive pole at the back of the neck, and moving the other along the side of the throat. Electrisation of the sympathetic has also been used as a method of treatment, but is probably as destitute of rational foundation as it certainly is of practical effect. Indeed, the result of all electrical treatment is most unsatisfactory. For an hour or two after each application there may be a little more power, but the effect does not last. I have never observed distinct evidence that electricity had the slightest influence on the course of the disease in any one of many cases in which I have seen it used.

In all cases the feeding of the patient demands much care. Semi-solid pulpy food can usually be swallowed better than liquids or solids. When deglutition is impossible without constant risk of the entrance of particles of food into the air-passages, and of prostrating and distressing paroxysms of choking, liquid food may be given by an œsophageal tube, or by means of a catheter or soft india-rubber tube passed through the nose into the upper part of the œsophagus. The food may be slowly poured into the tube through a small funnel, the patient's head being bent back. The only alternatives are the injection of food by the stomach-pump, or the less effectual method of rectal feeding with peptonised food. The larynx has been opened when the attacks of threatened suffocation were frequent and severe

Chronic Bulbar Paralysis without Anatomical Changes, anomalous and mysterious, has been occasionally met with. The cases have conformed, in general features and course, to the common degenerative variety, but have deviated from this in some minor point. Palsy of lips, masseters, and pharynx was attended, in one case,[*] with only slight weakness of the tongue, but with an amount of paralysis of the intercostals that caused fatal dyspnœa. No changes could be discovered in the nerve-nuclei, and the hypothesis of chronic peripheral neuritis naturally presents itself to the mind, but this was excluded by Oppenheim[†] in a case in which the larynx, pharynx, and tongue were paralysed. Degenerative changes in the lower part of the cerebro-nuclear fibres may have escaped detection.[‡] Moreover, function may be abolished by changes in the nutrition of the nerve-cells, that leave their aspect unaltered.

SUDDEN (APOPLECTIFORM) BULBAR PARALYSIS.

It has been already mentioned that various acute lesions of the medulla cause symptoms in the region of the nerves implicated in the degenerative form, and that these lesions generally differ from the latter in the less regular grouping of the symptoms produced. Occasionally a sudden lesion causes symptoms that are perfectly symmetrical, and correspond very closely to those of degeneration of the bulbar nuclei. Most of the sufferers have been advanced in life, —at the period in which arterial degeneration is common; but this form has also been met with in younger persons as a result of blows and falls on the head or neck,[§] or of syphilis,—influences which may cause disease in the vertebral artery, or lower end of the basilar, and narrow or occlude the branches. Such disease may cause successive sudden attacks, sometimes induced by depression of the general health or some influence that retards the circulation or promotes thrombosis. This is also true of another very rare mechanism, embolism, by which the vertebral may be closed (owing to the narrower calibre at its extremity), or its branches; the latter are commonly saved from separate occlusion by the angle at which they arise, which makes the entrance of a plug difficult, though not impossible. This arrangement, while it indisposes to embolism, promotes thrombosis. Among the many causes of thrombosis in this situation must be placed chronic basal meningitis and its effects on the arteries.[‖] The onset of the paralysis is sudden and "apoplectiform," sometimes with giddiness and vomiting, usually without loss of consciousness. The initial symptoms may be of wider range; there weakness of the limbs or an affection of sensibility, usually 'orm of subjective sensations, rarely actual anæsthesia. Any symptoms of a lesion of the part may co-exist, often only me; the wider symptoms pass away, and leave a permanent on closely resembling that of the degenerative form, but differ- the absence of a progressive tendency. For a time, indeed, ement may be conspicuous; the symptoms lessen, and then dition becomes stationary or nearly so, for a time which differs ie causal influences. As elsewhere, the symptoms may increase w days, owing to inflammatory processes in the vicinity of the fected area. Functional disturbance (as spasm in the masseters) en observed to precede or attend the onset. In some cases, time, degenerative changes supervene on the original acute and the case assumes a progressive character. Not seldom the ms, although bilateral, are unsymmetrical; but it is very rare m to be quite one-sided, as in a case recorded by Hirt, in which sis of one vocal cord and of the corresponding side of the tongue n suddenly; there was wasting of the tongue with the reaction neration.* Very often the symptoms, although bilateral, are r less irregular in distribution and character, as we should from the nature of their cause. This feature is often promi- nd important; there may be absolute inability to swallow, with alysis of the tongue or face. When the onset is by several ive attacks, each is usually slight, although one may be more than the others.

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[*] Dr. L. E. Shaw, 'Brain,' 1890.

[†] 'Virchow's Archiv,' Bd. cviii.

[‡] Compare vol. i, p. 440.

[§] As in the case of a boy aged twelve, recorded by Schulz ('Neurol. Centralbl.,' 1883, p. 99), in which bulbar paralysis followed immediately a blow on the back of the neck, and increased considerably a day or two afterwards. It is assumed that there was traumatic hæmorrhage, followed by secondary inflammation. Compare a case of fatal traumatic hæmorrhage recorded by Bochefontaine, 'Arch. de Phys.,' 1883, p. 160.

[‖] The possibility that thrombosis, found after death, may have been only a terminal event must also be borne in mind.

n example of this sudden form may be mentioned the case of aged sixty-three. At fifty-five he had a slight attack of right egia of ordinary type, without on of speech or of swallowing, which he recovered perfectly in urse of a few months. Five before being seen he suddenly e unable to articulate, and had lifficulty in swallowing. There o loss of consciousness. No e in his condition had occurred he came under observation. His ion then resembled perfectly of the progressive degenerative His lower lip hung down; constantly dribbled from the . The tongue was broad, flabby, lmost motionless, only the tip be brought over the lower teeth 'ig. 147). The palate was flaccid, but could be raised a little. wing was very difficult, and attacks of choking were frequent.

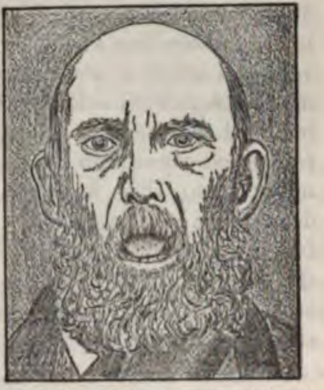

FIG. 147.—Bulbar paralysis of sudden onset, maximum protrusion of the tongue.

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* Hirt, 'Berlin. klin. Wochenschrift,' 1885, No. 26.

The vocal cords could be brought together, although with little force, so that no explosive cough was possible. He could still phonate, although with little modulation. There was not the slightest power of articulation; attempts to speak resulted only in "ah-ah-ah." Expression by writing was unimpaired. This patient was seen from time to time for five years after the onset, and his condition remained essentially unaltered. His power of swallowing varied; sometimes it became less, and then under treatment it improved again, but on the whole the loss of articulation and of swallowing, the paralysis of lips, tongue, and larynx, were neither better nor worse at the end of the five years than they were five weeks from the onset. There was no wasting and no loss of faradic irritability. As in this case, the muscles involved are usually those supplied from the medulla oblongata only, but a curious case has been recorded by Dixon Mann in which there was also some paralysis of the muscles of mastication.*

The pathology of these cases rests at present on little exact observation. It is probable that they depend on softening from vascular occlusion, situated in or near the middle line, and, at any rate in the cases with muscular wasting, damaging the nuclei that are the seat of degeneration in the progressive form. The position of the lesion in cases such as that detailed above, in which there is no wasting and no loss of electrical irritability, *i. e.* no evidence of damage to the nuclei themselves, is uncertain. It is probable that, in many cases, the lesion is just above the nuclei, so as to damage the paths from the cerebral hemispheres, which, after descending with the pyramidal fibres, certainly decussate near the nuclei, and would therefore be liable to common damage from a single lesion in the middle line. Thus, in a case described by Leyden, there was an area of softening in the middle line, at the level of the olivary bodies.† When the one vertebral artery is much smaller than its fellow, disease of the latter may damage both sides of the medulla.‡ But there is evidence that bilateral symptoms sometimes result from a one-sided lesion. The functional connection between the nuclei of the two sides is exceedingly close; they habitually act together in perfect equality, and destruction of those on one side may for a time interfere with the function of their fellows on the other side. In most cases of the kind the duration of life has been short, and it may be that the muscles of the other side would ultimately have recovered. No opportunity should be lost of ascertaining the exact nature of all cases of sudden onset, whether a microscopical investigation is undertaken or not.

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* Dixon Mann, 'Brain,' July, 1884, p. 244. It is very difficult to explain this direct combination of symptoms except on the hypothesis of a double lesion. But an association of indirect nature is conceivable, since the functions of chewing and swallowing are so constantly united.

† Leyden, 'Arch. f. Psych.,' vii, p. 44.

‡ The basilar may be, in effect, a continuation of one vertebral only, the other ending in the posterior inferior cerebellar artery.

The *Diagnosis* of these cases presents little difficulty. The symptoms are due to a sudden lesion of the medulla, and the cases in which they are symmetrical are not strictly separable from the others just mentioned, in which symptoms of similar character are of less regular and symmetrical arrangement. The distinction from the degenerative form rests on the mode of onset, sudden in the one case, gradual in the other. If a sudden increase of the paralysis should occur in the slow form, the preceding symptoms distinguish it from the acute variety. The nature of the lesion is shown by the indications that furnish guidance in other parts of the brain. It is important to note that symptoms may result from very slight vascular derangement, on account of the proximity of important parts, and that slight gradual derangement sometimes precedes the sudden attack.

The chief diagnostic difficulty in connection with these cases is due to the fact that the "pseudo-bulbar paralysis" mentioned on p. 320 may simulate very closely the symptoms of a sudden lesion of the medulla.* But, as a rule, the indications of a lesion in each cerebral hemisphere are clear. There are two distinct attacks, scarcely ever simultaneous, and it is the second attack that leaves the paralysis of the lips, tongue, and pharynx that simulates a lesion in the medulla. In most cases of the kind the disease has been in the central ganglia, especially in the lenticular nuclei; less commonly it has been in the white substance, or in the lower part of the motor cortex, or in this part on one side and in the central ganglia on the other. In cases of this nature, the diagnosis is not difficult if the observer is aware of the possibility of bulbar symptoms. A much greater difficulty exists if the bulbar symptoms follow a single apoplectiform seizure. In some instances, lesions occur simultaneously in both hemispheres; in others, a subsequent post-mortem examination has shown that only one hemisphere of the brain was diseased, and the lesion has sometimes been on one side, sometimes on the other. We have seen that an acute lesion may completely inhibit the function of the corresponding centre in the other hemisphere, even for days. It has been assumed that the lenticular nucleus has a special relation to the processes of articulation and deglutition,† an assumption which is scarcely consistent with the frequency with which it has been found diseased without the occurrence of bulbar (or other) symptoms. The implication of the internal capsule is generally possible in lesions of this nucleus. Another hypothesis is that in cases of one-sided lesion there is an exclusive relation of the affected functions to one hemisphere, owing to some congenital structural peculiarity.‡ On the other hand, it has been held that undetected microscopical lesions of the medulla are the real cause of the symptoms in these and many other cases of pseudo-bulbar paralysis.* The certain fact is that we have much still to learn by careful observation before these cases of unilateral disease can be comprehended.

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* Cases of this character have been described by T. Barlow, Joffroy, Lépine, Berger, Jolly, Ross, and others.

† Ross, 'Brain,' July, 1882.

‡ O. Berger, 'Breslauer Arztl. Zeitschr.,' 1884.

Besides the distinction often afforded by the mode of onset, pseudo-bulbar paralysis is characterised by the slighter degree of the symptoms, and by the facts that the nutrition of the tongue is unimpaired, reflex action is undiminished, and there is no change in electrical irritability. The larynx is seldom paralysed. These negative characteristics being sometimes present when the lesion is in the medulla, their diagnostic value is not absolute. The fact that a second lesion may occur in the medulla must also be borne in mind, and must occasionally lessen even the moderate amount of confidence with which the diagnosis of pseudo-bulbar paralysis can be made.

Prognosis.—Sudden apoplectiform bulbar paralysis involves much danger to life in the early stage of the disease, but the subsequent prognosis is less grave than in the chronic degenerative form. The tendency of the symptoms to progress is slight or absent. In many cases considerable improvement occurs; some recover entirely. In others, as in that described above, little or no power returns in the paralysed parts. The only guide as to the probable course of the symptoms is that afforded by the condition of the patient when the acute stage has passed. If, at the end of a month, there is no sign of improvement, and there is still a considerable degree of paralysis, it is improbable that much improvement will ensue. But the further forecast must depend on the nature of the lesion and on the indications available, which have been considered in the general account of such lesions. Then, if the evidence points, as it often does, to atheroma and thrombosis, the danger of a recurrence will be less, according to the absence of influences favouring thrombosis, or their limitation to such as may be obviated by suitable measures. The malady is only a local instance of the disease already considered.

The *Treatment* of this form illustrates the fact just mentioned; it is essentially that of acute softening from vascular occlusion, as described in detail at a previous page. The general management of the case, in regard to feeding, &c., is the same as in the degenerative variety.

ACUTE INFLAMMATORY BULBAR PARALYSIS

(POLIO-MYELITIS BULBI).

The term "acute bulbar paralysis" is commonly applied to the sudden form described in the last section, but is needed for a third variety, to which alone it is strictly applicable, in which the symptoms develop, not suddenly, in a few minutes, but acutely, in a few days. The mode of onset is that which is characteristic of acute inflammation, and that this is actually the lesion has been proved by several cases. In one observed by Etter,[*] a boy, aged fifteen, was taken ill with headache and vomiting, discomfort in the throat, difficulty in swallowing, and fever. In the course of the first week there developed bilateral paralysis of the face, accompanied by palsy of the tongue, greatest on the left side, paralysis of the palate, and of the left sixth nerve. Death resulted from pneumonia on the tenth day. The post-mortem and microscopical examinations revealed many myelitic foci in the medulla, symmetrical on the two sides, but greater on the left. They involved the left sixth nucleus, the left facial nerve within the pons, and the right facial nucleus, the left hypoglossal nucleus and the right hypoglossal fibres, and the accessory nucleus on each side, the motor part of the vagus nucleus on each side, and many cells and fibres in the tegmental region of the lower part of the pons. The foci of inflammation in the accessory nuclei could be traced down the cord as far as the fourth cervical nerves. Such a lesion is evidently closely analogous to that which, in the spinal cord, causes acute atrophic paralysis. We have seen (p. 194) that a similar inflammation may involve the nuclei of the ocular nerves in the upper part of the pons. The distribution in this case is that which has been found in the few others investigated, but the glosso-pharyngeal nuclei may also suffer. The inflammation is sometimes irregular in its distribution in the nuclei, and in one case it caused paralysis of all parts of the face. Hence this affection has been termed "acute inferior nuclear inflammation."

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* Oppenheim and Siemerling, 59th 'Versam. Deut. Aerzt.,' 1886.

Its causes are as little known as are those of the similar inflammation of grey matter in other parts.

The character of the changes seen with the microscope depends on the acuteness of the inflammation, varying from destruction of the nerve-elements and leucocytal aggregations or minute cavities of disintegration, to mere change of form and aspect of the nerve-cells, "cloudy swelling," loss of the processes, and ultimate shrinkage into small angular bodies. The increased interstitial tissue may present conspicuous spider-cells and the other features described in the account of analogous affections.

It is possible that similar symptoms are sometimes due to a peripheral neuritis of the bulbar nerves. Disease elsewhere prepares us for a similar susceptibility of the motor cells and their nerve-endings to morbid influences, which may act generally on one, occasionally on the other, owing to some slight difference in nature. But such acute peripheral bulbar neuritis has hitherto only been met with as part of a more general affection of the cranial nerves, such as existed in a case described by Eisenlohr,[†] in which there were hæmorrhages in nearly all the cranial nerves. Bulbar palsy was the predominant symptom, but included all parts of the face; the motor fifth was also weakened.

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[*] 'Corresp.-Bl. f. Schweizer Aerzte,' 1882, No. 24.

[†] 'Arch. f. Psych.,' ix.

The treatment of this affection must be that of other forms of acute inflammation of the brain.

ATROPHY OF THE BRAIN.

The whole or part of the brain may be below the normal size. In general atrophy the texture of the brain is normal; the whole brain is small, and the skull is also small (microcephaly). Little is known of the causes of this condition, whether the small size of the brain is the cause of the small size of the skull, or the reverse. The condition is generally associated with a high degree of mental defect of long duration, but an unexpected diminution in the size of one hemisphere is sometimes met with in chronic insanity, probably connected with a predisposition to the mental derangement.

In partial atrophy of the brain, one part is unduly small in proportion to the rest. The part so affected is usually changed in structure. It is firmer than normal—less firm only in rare cases of uncertain nature. It contains more connective tissue and fewer nerve-elements. Such sclerotic induration involves a difficulty as to the classification of the lesion, as already mentioned (p. 555). Almost any part of the brain may be thus smaller than normal—the whole of one cerebral hemisphere, or only part of it, seldom both hemispheres, the central ganglia on one side, the pons, or the cerebellum. Atrophy of the cerebellum may involve the whole or only one hemisphere, or both hemispheres—the middle lobe being normal. When the whole of one cerebral hemisphere is atrophied, one part is usually more affected than the rest. Several parts of the brain are frequently atrophied together, in a manner that shows a causal relation, depending on a structural connection, in consequence of which the atrophy of one part entails that of the other. Thus atrophy of the whole of one cerebral hemisphere is usually associated with atrophy of the opposite cerebellar hemisphere. The latter may be associated with atrophy of the opposite corpus striatum without the rest of the hemisphere, and it is usually associated also with atrophy of the opposite olivary body of the medulla oblongata. The parts concerned in vision may be thus marked out by separate wasting (see p. 71). The arteries may share the diminution in size without presenting any special disease.

Partial atrophy of the brain is probably in most cases acquired. This is clear in many instances in which it is attended with symptoms that commenced during the first years of life. In some of these cases the lesion dates from birth, and the change in the brain is the result of its compression by local meningeal hæmorrhage (see p. 413). In other instances the lesion occurs during infancy, and is accompanied by "infantile hemiplegia," in the account of which (p. 456) will be found some evidence as to the nature of the disease. In some cases, again, it is probable that an attack of meningitis was the cause. Both hemispheres may suffer from this cause, and also in the cases of meningeal hæmorrhage during birth. In all varieties, the local atrophy is often accompanied by a general diminution in size of the hemisphere, no doubt due to the connection that exists between all parts, and to the effect of considerable damage to one part of the growing brain on the development of the rest. With only local atrophy of the superior parietal lôbule, one hemisphere of an adult has been found one and a half inches shorter than the other.*

Partial atrophy of the brain may also develop during intra-uterine life. It may be found present at the time of birth. In some cases it is perhaps due to intra-uterine disease similar in character to that which, after birth, has similar consequences. More frequently the atrophy is such that it can scarcely be thus explained. Atrophy of the cerebellum is more frequent in these cases than is atrophy of one cerebral hemisphere. Sometimes both cerebellar hemispheres are very small, and the middle lobe is of normal size. It is very difficult to explain this condition on the supposition of any intra-uterine morbid process; it would seem rather to be connected with the entire absence of the cerebellum sometimes observed. This is apparently due to some perversion or defect of the process of development; the cause of this is practically unknown, but we are probably safe in ascribing it to a positive germinal defect.

The symptoms that attend atrophy of the brain vary much. Mental defect is the most constant, and usually amounts to idiocy. Hemiplegia and epileptic fits often accompany atrophy of one cerebral hemisphere. Bilateral weakness, with athetoid movements and inco-ordination, may attend bilateral atrophy, such as is due to difficult birth and meningeal hæmorrhage compressing the cortex. In many cases, however, it is certainly incorrect to regard these symptoms as the direct consequences of the diminished size of the convolutions; they are the result of the disease which has also caused the atrophy,—the local atrophy directly, the slighter general atrophy of the hemisphere, which is often present, indirectly, by checking its growth. In atrophy of the whole of the cerebellum, unsteadiness has been observed similar to that which results from disease of the middle lobe. When only the hemispheres have been affected, there have sometimes been no motor symptoms, but in some cases there was intellectual defect. Atrophy of one hemisphere of the cerebellum has also been found when no symptoms, that could be ascribed to it, were present during life.

Senile Atrophy.—In old age the brain wastes, like many other organs, and becomes smaller and firmer. The amount of fluid in the ventricles and on the surface is increased in proportion to the lessened bulk of the brain. This natural increase of fluid was formerly regarded as pathological, and a mysterious death was ascribed to "serous apoplexy"—a purely imaginary lesion, not yet quite forgotten. But this wasting of the brain is commonly attended by no symptoms. Senile mental failure is often ascribed to it, but, since it may exist in considerable degree without the slightest mental defect, caution should be observed in attributing to it any mental change that may co-exist.

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* Fraser, 'Glasgow Med. Journ.,' 1889.

HYPERTROPHY OF THE BRAIN.

Under the name "hypertrophy of the brain" a condition has been described in which the brain is of abnormal size. Sometimes it has been found also to be of distinctly abnormal weight, but the wide variations in the weight of the brain under normal conditions render this criterion decisive only in extreme cases.* The size of the brain in relation to the size of the skull is the feature that has attracted most attention. If the condition develops before the bones have united, the skull is said to undergo enlargement similar to that of hydrocephalus. If the sutures have been closed, the convolutions are compressed and pale; and when the brain has been removed, it cannot be replaced in the cranial cavity—a feature which, since the days of Morgagni, has attracted attention, perhaps more than it deserves. In all cases the ventricles are empty; their walls are pressed together, and the vessels of the brain contain little blood.

Decisive examples of this state are extremely rare; there are few satisfactory observations on the minute structure of the enlarged organ, and we do not therefore know whether this is the same in all cases. In some, an increase in the neuroglia has been found, and has been regarded as the cause of the enlargement (Virchow; Tuke, in an unusual case in which the enlargement was confined to one hemisphere). In other cases no increase in the connective tissue could be found.

Such enlargement of the brain has been met with chiefly under three conditions. (1) In very young children soon after birth. It is in these that the hydrocephaloid enlargement of the head has been described. (2) Towards the end of the first year of life, in association with rickets. It may be that, the closure of the skull being retarded, the brain attains an abnormal size in consequence of the deficiency of the mechanical restraint that should be furnished by the skull when its bones unite. It has been thought that the enlargement of the brain increases the size of the skull, but the shape of this is that characteristic of rickets, and the size may be due to the double influence of abnormal ossification, and the enlargement of the brain. (3) An increase in size has been occasionally observed in older children and even in adults, but of its nature very little is known.

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* The weight and size of the brain differ according to age and stature, but even thus tested the variations are great. Proportion to absolute body-weight has also been taken as a guide, but it is not to be relied on, since the body-weight varies in each direction, that of obesity and emaciation, irrespective of stature. If the body-weight is taken as a term of comparison it should be the average for each age and stature

Of the causes of this condition only two facts can be regarded as established: first, that it has been occasionally observed in families; secondly, the relation to rickets already described.

The symptoms that it produces are very uncertain. The form that occurs soon after birth and causes enlargement of the head is said to be attended by nervous symptoms almost identical with those of chronic hydrocephalus, so that the distinction of the two is practically impossible. It is doubtful whether the rickety enlargement causes any symptoms. We are not justified in referring to it the laryngeal spasm or general convulsions or mental backwardness of rickety children, since these may be present when there is no enlargement of the brain.

Even more doubt exists as to the symptoms of the form that has been met with at a later age. In some cases it has apparently caused none. In others, acute cerebral symptoms, resembling meningitis, have occurred, and have run a rapid course, ending in death. The evidence of a connection between the enlargement of the brain, which was regarded as chronic, and the acute symptoms is not satisfactory, and no explanation of the supposed connection has been attempted. The condition seems never to have been recognised during life, and nothing is known of its treatment.

The so-called hypertrophy of a small part of the brain, as of certain convolutions, or of the pons Varolii, has probably been always due to an infiltrating growth (see p. 495).

From what has been said, it is clear that the pathology of enlargement of the brain needs fresh study in the light of future investigation. Moreover, the common tendency to speak of the enlargement caused by overgrowth of connective tissue as "hypertrophy" of an organ, is an inexact use of the word, which, when applied to an organ without qualification, should designate only an enlargement chiefly due to an overgrowth of the elements on which the function of the organ depends.

HYDROCEPHALUS.

Hydrocephalus, or dropsy of the brain, consists in an accumulation of fluid within the skull, either in the subdural space (external hydrocephalus) or within the ventricles (internal hydrocephalus). It may be acute or chronic, the result of other morbid processes that are conspicuous ("secondary form"), or the consequence of a process that is chiefly manifested by this effect (so-called "primary form").

Acute Hydrocephalus.

The only known cause of actually acute hydrocephalus is meningitis. This, in all forms, may be attended by effusion of fluid in the subdural space or into the ventricles. The latter, for instance, occurs in four fifths of the cases of tubercular meningitis. Hence the term "acute hydrocephalus" was for a long time the common designation for meningitis. The external effusion is the direct result of the inflammation of the pia mater, and the internal effusion is probably also the result of inflammation of the choroid plexuses and velum interpositum, perhaps also of the lining membrane of the ventricle.

In some cases, however, the effusion into the ventricles is the only pathological change in an affection of acute and febrile course. The external meninges are healthy; the choroid plexuses may be the seat of distinct inflammatory changes, and the lining membrane of the ventricles may be finely granular; the adjacent brain tissue is softened, and the brain-substance and convolutions are compressed. There is no indication of any processes of which the effusion could be a mechanical consequence, such as is an occasional cause of chronic hydrocephalus. The symptoms during life are those of an acute inflammatory disease, and resemble very closely those of tubercular meningitis. Hence it is commonly assumed that they are due to a ventricular meningitis, affecting chiefly the choroid plexuses, and causing an abundant effusion of serum. Although the pathology of the cases is obscure, no better explanation has yet been given.

Chronic Hydrocephalus.

External Chronic Hydrocephalus.—Whenever there is wasting of the brain, there is increase of the subarachnoid fluid to occupy the space left. This is a common senile condition. Of the same character is the accumulation of fluid met with in some cases of arrested development, in which the brain is small and does not fill the cranial cavity.

In other cases, without the brain being smaller than normal, there is a congenital excess in the amount of fluid in the subdural space, and this expands the skull. The expansion may be so great as to prevent the child being born alive. If birth is survived, the head rapidly increases in size, and is of the same shape as in the internal hydrocephalus, to be immediately described. Post mortem, the excess of fluid is the only morbid condition. There is no change in the membranes, and the origin of the condition is not known. It is usually attended by symptoms similar to those of internal effusion, having a similar course. Caries of the bone of the skull, in such cases, has occasionally permitted the escape of the effusion.

External hydrocephalus is sometimes "sacculated," *i. e.* limited to a certain region by adhesions between the dura and pia mater. This condition is, in effect, a meningeal cyst. Its position may be over part of one cerebral hemisphere or beneath the tentorium. If there is enlargement of the skull, this is correspondingly unsymmetrical. The brain may be compressed opposite the seat of the effusion, and corresponding local symptoms may result. These cases doubtless arise from inflammation, and their course may be varied by intercurrent attacks of a meningitic character.

CHRONIC INTERNAL HYDROCEPHALUS may be either congenital or acquired.

Congenital Internal Hydrocephalus develops *in utero*, and may cause such enlargement of the head as to prevent birth until the fluid is let out; or it may be moderate at the time of birth, and afterwards rapidly increase. The causes and pathological mechanism are practically unknown. It has been ascribed to maternal grief or other emotion, but on no reasonable grounds, and also to injury of the fœtus by falls, &c.,—an influence which is at least intelligible. A connection has been supposed to exist between uterine disease and hydrocephalus, and has been explained hypothetically, but it has yet to be proved. There is, however, no doubt that a tendency to the occurrence of fœtal hydrocephalus sometimes runs in families, and that more than one child may be affected in succession.

Sometimes all the ventricles are distended; more often the fourth ventricle suffers little, even when there is no obstruction in the aqueduct of Sylvius. In such a case the aqueduct may be funnel-shaped, dilated towards the enlarged third ventricle. If the distension is confined to one or both lateral ventricles, there is some obstruction at the foramen of Monro (see further, under "Acquired Hydrocephalus"). The fluid is usually clear, of low specific gravity, 1001—1009, and contains a small and variable amount of albumen, some chloride of sodium, and sometimes urea, cholesterin, and other substances in small quantity. The amount of liquid varies according to the degree of the disease, and has been as much as twenty-seven pounds. In proportion to its quantity, the substance of the cerebral hemispheres is compressed, and thinned by extension and wasting, especially the white substance, and, it is said, the callosal fibres.* The corpus callosum is displaced upwards, and if the cranium is enlarged, the hemisphere extends further forwards, backwards, upwards, and outwards than normal. The cerebral substance may be reduced to a layer only a few millimetres thick, constituting the thin wall of a vast cavity. In such a case, all traces of the convolutions and sulci may have disappeared, and even the basal ganglia may be almost unrecognisable. More often indications of the sulci can be traced, and the compressed basal ganglia lie at the bottom of the sac. The cranium is enlarged in proportion to the amount of effusion, and the bones of the skull are thin. The falx is necessarily stretched, and its edge forms a larger curve than normal; the extension of its edge raises the anterior part of the tentorium. Hence the subtentorial space is increased in size, and is not completely filled by the cerebellum, the interval being occcupied by liquid, and sometimes by loose connective tissue. Otherwise the membranes are normal. The choroid plexus may be thickened, as if from old inflammation. The lining membrane of the ventricles is often finely granular on the surface, and is sometimes thickened.

The external enlargement of the skull is very conspicuous and characteristic, and rapidly increases after birth. The fontanelles become very large, and bulge; at the sutures, the bones are widely separated. The cranium has a rounded shape, and becomes disproportionately large in comparison with the face. The disparity is increased by the projection of the frontal portion of the skull. The orbital plates have an oblique direction, and the eyes are directed downwards and partially covered by the lower eyelids. If the child lives, the head may attain an enormous size; in one case, at sixteen months old, the circumference was 107·6 cm. (Klein). The symptoms vary much. There is usually considerable mental defect, often amounting to idiocy. The limbs are weak; convulsions and various contractures are frequent; occasionally febrile attacks occur, attended by vomiting. The head is supported with difficulty, on account both of its weight and of the muscular weakness. The eyeballs are often rolled from side to side, and their axes may not correspond. The skin of the head is thin, and the hair scanty. In extreme cases, blindness occurs, and the ophthalmoscope shows optic nerve atrophy, produced by the stretching of the nerve, or by the compression of the chiasma. In congenital cases, the rapid increase of the disease usually causes death in the second or third month of life, by marasmus, convulsions, or coma. Occasionally the morbid process becomes arrested, and the patient may live on to adult life and even to old age. It is said that the fluid is sometimes gradually absorbed. Ossification of the bones may progress, and is usually completed by the development of "Wormian bones" within the sutures. If the disease is considerable in degree, the mental and motor symptoms usually continue in some degree through life—mental weakness, often with irritability of temper, epileptic fits, muscular weakness, and contractures.

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* Anton, 'Wien. med. Jahrb.,' 1888.

Acquired Chronic Internal Hydrocephalus may be (1) secondary to a lesion that produces the effusion mechanically; (2) consecutive to an attack of meningitis; (3) of apparently primary origin.

Secondary Mechanical Form.—Whytt pointed out in the last century that any obstruction of the veins of Galen, hindering the return of blood from the intra-ventricular vessels, causes effusion into the ventricles, which may reach an extreme degree. The most common cause is an adjacent tumour. But an obstruction to the passage of fluid out of the ventricles is another mechanical cause, commonly co-existing with the pressure on the veins, and more certainly effective when it acts alone. The fluid normally escapes by the openings in the membrane closing the fourth ventricle, the foramen of Magendie in the middle line, and the openings, one on each side, behind the roots of the glosso-pharyngeal nerve, first described by Mierzejewski. These may be occluded by meningitis, and then all the ventricles become distended; the passage from the third to the fourth ventricles is closed, only the ventricles above are distended, and from obstruction at the foramen of Monro the effusion is confined to the lateral ventricles. Progressive effusion is probably in most cases the result of one of the two mechanical processes just mentioned, chiefly of the hindered exit (see Hilton on "Rest and Pain"). A moderate amount of effusion may be associated with diseases (as abscess or tumour) which do not involve any distinct mechanical influence. Its mechanism is obscure.

Primary Hydrocephalus.—Internal effusion sometimes occurs without any of the above causes being traceable. In children with yielding skulls, it is supposed that the mechanical congestion of frequent cough, or the tendency to transudation present in anæmia, may lead to some effusion, but it is not probable that the amount from these causes is ever considerable. At any age, the condition has been known to develop and attain a considerable degree, without, it is said, any other pathological condition being discoverable than the slight traces of ventricular inflammation that are met with in the congenital cases. Dean Swift died from this disease at seventy-eight, after it had existed for three years. These cases are probably due to obstruction.

The symptoms in the acquired form are, on the whole, similar to those in the congenital variety, due allowance being made for differences of age. These are mental weakness, somnolence, coma, muscular weakness, convulsion, contractures, and loss of sight from pressure of the distended third ventricle on the optic chiasma. Temporal hemianopia may, therefore, precede the complete blindness. In young children, with incomplete closure of the sutures, the head readily enlarges, although rarely to the same extent as in the congenital form. In the adult, enlargement of the head is less common, although it has been known to occur, and the sutures may, as I have seen, become separated, usually after the gradual thinning of the cranial bones described in the section on tumours of the brain (p. 503). It might be supposed that the intra-ocular circulation would show the effects of the increased pressure within the skull, but this is seldom the case, on account of the anastomoses of the ophthalmic vein. Even during the process of separation of the sutures, I have been unable to observe any marked increase in the size of the retinal veins. The course of the acquired form varies much. Death usually occurs at the end of a few months or years. The progress sometimes ceases, and if the affection is moderate in degree, recovery may occur. It is only in children, in whom the ready enlargement of the head enables a certain diagnosis to be made when the disease is still in an early stage, that recovery can be proved. The sac has been known, in very rare instances, to rupture into the subdural space.

Diagnosis.—As just intimated, hydrocephalus can only be diagnosed with certainty when there is distinct progressive enlargement of the head. When the bones are united, internal effusion may be suspected if its indications slowly follow an attack of meningitic character, or accompany the symptoms of a tumour of the cerebellum. But primary hydrocephalus causes only symptoms that are not distinctive, and are much more frequently produced by other morbid processes. Hence, as a matter of fact, the existence of the disease, when it leads to no enlargement of the head, and when the causal indications just mentioned are absent, cannot be recognised.

In slight degree the enlargement of the head may be confounded with that produced by two other causes, rickets and thickening of the bone. In the former the head has a somewhat square form, and not the globular shape characteristic of hydrocephalus. Although the fontanelle may be large, it is not bulged. The other signs of rickets are present in high degree. Thickening of the cranial bones may simulate hydrocephalus at almost any age. I have seen it in a boy of ten, the subject of inherited syphilis, and in a man of fifty. In the latter, a slow progressive enlargement of the head during several years led to a diagnosis of hydrocephalus, but at the post-mortem examination the cranial bones were found to be three quarters of an inch in thickness, and the cavity was of normal size. It is doubtful whether the nature of these rare cases can be ascertained during life. A distinction of internal from external hydrocephalus can only be made by paracentesis, and not always with certainty even by this means, since a thin-walled sac may be readily opened by a comparatively superficial puncture.

The Prognosis of hydrocephalus of any form is usually grave and always uncertain, unless the occurrence of arrest can be distinctly recognised.

TREATMENT.—Whatever lessens the volume of the blood diminishes for a time the amount of the effusion. Thus an attack of diarrhœa lessens the prominence of the fontanelle. But purgatives are inadmissible; to be effective they must be more vigorous than a hydrocephalic child can bear. Diuresis constitutes a safer, but unfortunately less effective, mode of attaining the same end. In no case in which these measures have been used has a permanent effect been produced. Agents that are supposed to promote absorption, as iodide of potassium and mercury, have been extensively tried, but are as a rule powerless, and are sometimes harmful. The most direct treatment, which is unfortunately the most dangerous, is evacuation by puncture with a trocar or aspirator, a small quantity being let out each time, and compression of the skull by elastic bandages kept up during and after the operation. This procedure is of course most suitable to external hydrocephalus, but it has been employed in ventricular effusion, occasionally without ill effects, but with absolute success only in rare instances. The place for puncture was formerly at the outer angle of the anterior fontanelle, but antiseptic measures have permitted trephining and puncture in a locality at which the lateral ventricle is most accessible. Continuous drainage has even been employed, but the result has seldom been satisfactory, whatever the nature of the case.* The first puncture has generally been well borne, and has relieved urgent symptoms of cerebral compression, but the second or third has usually had little effect, and has sometimes been fatal—in some instances apparently because too little time was allowed to elapse between the operations. Puncture of the spinal membranes below the cord has also been resorted to. It can, of course, relieve internal hydrocephalus only when there is no obstruction to the passage of the fluid from the ventricles, and this the operation only may decide. It has relieved urgent symptoms for a time, and has been thought capable of starting absorption in chronic cases, but its demonstrated service is limited to relief. On the other hand, it seems to do no harm.†

Good has been observed to follow simple compression of the skull, a mode of treatment first advocated and energetically employed fifty years ago by Barnard, of Bath. Trousseau employed strips of diachylon plaster, a third of an inch broad, in the following manner:‡—(1) From each mastoid process to the outer part of the orbit on the opposite side; (2) from the hair at the back of the neck, along the sagittal suture, to the root of the nose; (3) over the whole head in such a manner that the different strips shall cross each other at the vertex; (4) a long strip around the head three times, taken first above the ears and eyebrows, and a little below the occipital protuberance, so that the ends of all the other strips shall project below the circular strip; these ends are next to be doubled up on the circular strip, and its remaining two turns passed over them in the same direction as the first turn. It is necessary to watch the effect, and loosen the strips if there are any symptoms of compression. Dr. West advises a broad elastic band as safer and more manageable than the plasters. His experience of pressure in severe cases has not been encouraging, since it has not hindered the accumulation of fluid, and has increased the symptoms of compression of the brain.

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* See Keen, 'Phil. Med. News,' 1889, and 'Trans. Tenth Int. Med. Congress;' Robson, 'Brit. Med. Journ.,' 1890.

† See Quincke, 'Berlin. klin. Wochenschrift,' 1891, for the details of ten cases, five of whom were adults; also Wynter, 'Lancet,' 1891, i.

‡ 'Journal de Médecine,' April, 1843, quoted by West, 'Dis. of Infancy and Childhood,' 7th ed., 1884, p. 136.

PART V.

GENERAL AND FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

THE diseases that remain for consideration are those in which there are no constant changes to be seen with the naked eye. It was formerly the custom to include them all under the term "functional diseases," but microscopical changes have been discovered in some of them with sufficient frequency to make it certain that there is far more than a mere disturbance of function, and it cannot be doubted that most of these maladies depend upon alterations in the nutrition of the nerve-elements, although these may not yet have been found, and perhaps cannot be detected without more means of investigation than we at present possess. The diseases themselves are so different in character and so various in seat, that their classification into groups is alike difficult and useless. Anything like a scientific classification, based upon our present knowledge, would result in little more than enumeration, and is therefore not attempted. The order in which they are described is based only on convenience.

CHOREA.

Chorea is a disease that occurs chiefly in young persons, is usually of limited duration, and is characterised by irregular spasmodic movements, by inco-ordination of voluntary movement, and often by muscular and mental weakness. The proportion of these elements varies in different cases.

The term "Chorea Sancti Viti" (χορεία = dancing), or St. Vitus's dance, was first applied at Strasburg to the epidemic dancing mania prevalent in the fourteenth and fifteenth centuries, when the sufferers were taken, by order of the Strasburg magistrate, to the chapel of St. Vitus, to be cured by the influence of the saint. The term became restricted, chiefly through the influence of Sydenham, to the disease now known by the name, but it is even now used more widely in Germany, and the affection here described is distinguished, as *chorea minor*, from *chorea major*, a form of hysterical disorder distinct from ordinary chorea, and more closely allied to the epidemic malady of the Middle Ages.

Etiology.—The relation of chorea to *race* is a subject that has been but little studied. According to Weir Mitchell, in the United States the disease is less frequent among the negro than among the white population, and in some places in which there is a mixed population (*e. g.* in the island of Cuba), where chorea is not uncommon among the white children, it is unknown among the coloured children. Urban residence is said by the same writer to favour the occurrence of the disease, and it is probable that the influence could be traced also in this country.

Hereditary influences can be found in many cases of chorea, and a double relation of the disease can often be traced in these influences; its relation to acute rheumatism on the one hand, and to other nervous diseases on the other. These relations may be separate or may be combined. Rheumatism is so common that it is only significant when the family tendency is strong or close, unless, as is frequently the case, the patient has also suffered from rheumatism; it is, however, sometimes very pronounced, and some family tendency can be traced in 45 per cent.* A neuropathic heredity has been found in one sixth of the cases—epilepsy, insanity, or chorea itself. This proportion is not great, but the actual facts, in the case of neuroses, always exceed those that can be ascertained, and often much exceed them. Moreover they are emphasised by the strong character of many instances. For example, three sisters suffered from true chorea; a sister also suffered from chorea, and the mother from epilepsy; the mother of one patient was insane, and her sister's child also had chorea; the father of another had suffered from chorea in early life, and so had two children of his brother; five relations of another had been insane. Many other similar facts have been met with in the investigation of the clinical history of epilepsy. For instance, a man was epileptic, his sister insane, and two of her children had chorea. Two sisters of an epileptic girl had had chorea. Two children suffered from chorea, their mother's sister being epileptic and insane.†

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* Brit. Med. Association's Collection (see note on next page).

† Many illustrations of a double family tendency are mentioned by Money in his analysis of 236 cases of chorea contained in the case-books of University College Hospital and of the Hospital for Sick Children ('Brain,' 1882, vol. v, p. 513). In two cases the patient's father had suffered from rheumatic fever and chorea; in another both the father and the mother had had rheumatic fever, and a brother

Age.—Chorea is essentially a disease of the later period of childhood. Nine tenths of the cases occur between 5 and 20, and four fifths between 5 and 15. A larger number of first attacks (nearly half the total) occur between 5 and 10 than between 10 and 15; but according to my own observations, if relapses are included, more cases occur between 10 and 15, and the thirteenth year of life is that which presents the largest number of attacks.* Under 5 the disease is extremely rare; a few cases have been recorded at 4, and one recorded case (with endocarditis) in a girl commenced at the age of 2 years 11 months.† Not more than 5 per cent. of the total number of attacks occur over 20. The disease is, however, occasionally met with in later life, even up to extreme old age. Between 30 and 40 the disease is scarcely ever met with. Many cases of senile chorea probably depend on a morbid process different in nature from that which causes the juvenile form of the disease, although similar in effect.

Sex.—Chorea affects girls nearly, but not quite, three times as frequently as boys. A combination of recorded statistics yields 365 boys to 1000 girls.‡ The preponderance of girls is least in childhood, and increases after puberty. The disease is rare in lads over 16. Between 20 and 30 it is practically confined to females. During the second half of life, however, the rare cases of chorea occur in both sexes, and seem a distinct variety.

Climate has little influence. Chorea occurs in warm as well as in temperate countries. The question of its relation to *season* has recently attracted attention, especially in America. At Philadelphia, Morris Lewis found an apparently distinct relation to time of year,

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chorea; in another the mother had had rheumatic fever, and mother, father, and brother had had chorea. Great variations exist in the proportions of both rheumatic and neurotic heredity given in the collection of cases made by different observers. That of the former has been thought to be due to locality, but both are probably chiefly the result of the narrowness or width of the conceptions applied to the facts ascertained.

The special form of "hereditary chorea" (affecting many generations) is separately described on a later page.

* The 439 cases tabulated by Dr. S. Mackenzie for the Brit. Med. Assoc. Collective Investigation Committee ('Brit. Med. Journal,' February 26th, 1887) give 34 per cent. (one third) between five and ten, 43 per cent. between ten and fifteen, and 16 per cent. between fifteen and twenty; but unfortunately it is not stated how many were first attacks. In this list are cases at forty, sixty-three, sixty-eight, seventy-three, seventy-eight, and eighty-six. The facts there stated agree closely with those in the text, which have been furnished by an extensive and unselected series of cases that have come under my own observation.

† Sturges, 'Lancet,' 1888.

‡ The statement in the text is based on the cases collected by Hughes, Sée, Pye-Smith, Rufz, Steiner, Wilkinson, Sturges, and on 100 original cases. Correct facts regarding the influence of age can only be ascertained by combining statistics, since they are not fairly represented either by the figures obtained from Children's Hospitals, or by those from General Hospitals in towns in which Children's Hospitals exist.

most cases occurring in the spring months;[*] in Boston, however, Putnam failed to trace such an influence, and in this country the effect of season does not seem to be great, the numbers (of 100 attacks) commencing in each quarter being—1st, thirty-three; 2nd, twenty-five; 3rd, twenty; 4th, twenty-seven. The minimum was in July and August. A more distinct relation to season is, however, occasionally to be traced in the recurrences of the disease (see p. 608). An occasional special frequency of the malady is often to be observed, perhaps connected with the influence of weather, but definite proof of this is wanting. In its study, the cases with neurotic and with rheumatic heredity should be separated; this influence may be true of the latter only, and obscured if all are taken together.[†]

The only immediate cause of chorea that can be traced with any frequency is emotion, usually fright, rarely mental distress. The proportion of cases in which mental emotion can be traced has varied in different collections, between a fifth and a fourth. I have found the frequency to be nearly the same in each sex, but it is relatively more common in boys under twelve, and is rare in boys over fourteen, whereas it is a not uncommon excitant of chorea in girls up to twenty. The interval between the fright and the first symptoms of the chorea rarely exceeds a week;[‡] it is about as frequent for it to be one week as to be less than a week (three or five days). Very rarely the interval is only one day, and still more rarely there is no interval,—the chorea immediately succeeds the fright. This was the case in a lad in whom the movements commenced immediately after a pistol had been unexpectedly discharged close to his ear. Another boy was discovered in an apple tree, and fell in his hasty descent; he immediately began to shake; the tremor of alarm assumed a choreic character, and persisted as chorea.

Occasionally the fright, which was the cause of the chorea in girls, occurred at the menstrual period, but such instances are too rare for any special significance to be attached to the coincidence. Nor is any disorder of menstruation to be recognised with sufficient frequency to justify the assumption of a causal influence. Imitation is often said to be an occasional cause of chorea; the hysterical variety often arises thus, but it is doubtful whether true ordinary chorea is so produced. It should be noted, however, that the connection between some cases of apparently hysterical chorea and the common form is exceedingly close. In the epidemics of chorea that are occasionally met with among children at the same school, for instance, most cases are distinctly of the hysterical variety, but in a few the movements may resemble those of the common form.

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[*] The percentage varied from 4·1 per cent. in October, and 4·3 per cent. in November, to 8·2 per cent. in January, 15·3 in March, 8·6 in April, 10·7 in May, and 10·5 in July, and then fell gradually to its lowest point in October.

[†] Dr. Morris Lewis instituted an elaborate inquiry with the object of ascertaining whether there is any relation between temperature, humidity, or barometric variations and the occurrence of chorea (compared over a period of ten years), but he could find none. He found, however, that there is a slight correspondence with the average number of cloudy days per month, and also with the actual number of rainy days, and a still closer connection with the number of storm centres passing over Philadelphia; and that the correspondence became closer the wider the range of country included in the meteorological observations, till an area of a radius of 400 miles was reached, and then the correspondence between the storm curve and the chorea curve became still more close. Comparing the average number of attacks per month of chorea and rheumatism, the curious fact was ascertained that the variations in the occurrence of rheumatism corresponded with those of chorea, but were uniformly a month later. (The 'Polyclinic,' Jan., 1887.)

[‡] In the cases in which many weeks elapse between the fright and the first symptoms, a causal relation is extremely doubtful.

Chorea sometimes results from traumatic influences, but it is probable that their efficiency depends on coincident emotion. Thus chorea has resulted from a fall or a blow on the head and from the extraction of a tooth, but it has also followed an attempt at tooth-extraction which was unsuccessful.

Intestinal worms are occasionally present in choreic patients, and have been supposed to cause the disease. In extremely rare cases chorea has been ascribed to the irritation of a peripheral spinal nerve. The influence of these reflex causes is doubtful, since they are so seldom met with, but it can scarcely be denied.

Chorea is so rare in association with acute specific diseases, that such a coincidence is probably accidental; or, at most, the general disease only serves to excite the chorea in an indirect way. A girl sixteen years old had a well-marked attack of chorea during the course of typhoid, but she had suffered from acute articular rheumatism a year before.* Toxic influences may perhaps sometimes cause it. An attack and relapse seemed clearly the result of the absorption of iodine in one case; in another, severe coincident multiple neuritis suggested a blood-state of peculiar character.†

Three important etiological influences remain for discussion, two of which have given rise to much controversy,—acute rheumatism, organic heart disease, and pregnancy.

Acute Rheumatism.—The fact that a considerable number of patients with chorea have had acute rheumatism has been recognised by all observers since it was first pointed out by Hughes and by G. Sée, although the extent and significance of the association have been variously estimated. The largest recorded proportion of cases with preceding rheumatism is one half.‡ In my own cases the proportion was about one fourth, but varied at different ages; in only one case, under nine, was there a history of rheumatism, while of 53 cases which occurred between ten and fifteen years, 16, considerably more than one fourth, had suffered from rheumatic fever. This fact shows clearly that the influence of rheumatism will be under-represented in statistics collected at hospitals for children. The proportion, in later childhood at least, is too large to be ascribed to accidental coincidence, and the close relationship between the two diseases is shown in an emphatic manner, by the facts that not very rarely chorea immediately follows acute rheumatism, that the latter occasionally comes on in the course of chorea, and also by the mysterious but unquestionable relation next to be mentioned between chorea and disease of the cardiac valves.

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* Peiper, 'Deut. med. Wochenschrift,' 1885, No. 8.

† Fry, 'Journ. Nerv. and Ment. Dis.,' 1890.

‡ Tylden, 'St. Bart. Hosp. Rep.,' found some rheumatic history, family or personal, in 72 per cent. About a fourth is the proportion found by Hughes, two fifths by Sée, and a fourth by the 'B. M. A. Coll. Invest. Rep.' (loc. cit., p. 428). In the latter the percentage was 26, and about the same in each sex; but when to these cases are added those in which acute rheumatism occurred during or after the chorea, the percentage rises to 32, or one third, and another

Heart disease may be developed during the course of chorea, or may precede it. The latter only concerns us now. Published statistics afford no information on this point, for no attempt has been made to discriminate between the two forms.* When the cardiac disease is considerable in amount, and the chorea of short duration, and especially if the walls of the heart present evidence of consecutive changes, we are justified in assuming that the heart disease preceded the chorea, and this assumption is rendered the more probable if there has been a preceding attack of rheumatic fever. Of forty cases in which I found evidence of organic heart disease there was strong reason to believe that this preceded the chorea in eighteen. From the nature of the investigation, this proportion is probably below the actual facts (because only conclusive evidence was admitted), and it is therefore probable that in about one half of the cases in which organic disease of the heart exists, this preceded the attack of chorea in which it was evident.

Regarding the combinations of the three conditions, rheumatism, organic heart disease, and fright, I have found that cardiac disease was present (either before or during the chorea) in three quarters of the cases in which there was a history of rheumatic fever, while in those without such history there was heart disease in only a third. Fright was the apparent cause of the chorea in a rather larger proportion of the patients who had had rheumatic fever, or preceding heart disease, than of those who had not.

Pregnancy.—Chorea may occur as a complication and consequence of pregnancy. The hysterical form, to be presently described, is sometimes met with, but as a rule the malady corresponds with the form met with in early life. Some of the patients who thus suffer have had chorea or rheumatic fever in childhood or youth,† or an

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14 per cent. had suffered from vague rheumatic pains. For other statistics see Herringham, 'Med.-Chir. Trans.,' vol. lxxii, and Syers, 'Lancet,' 1889.

* To this statement, however, the 'B. M. A. Collect. Inv. Report' constitutes an exception.

† Of course the converse must not be assumed; very few of those who have chorea in childhood suffer from it during pregnancy.

attack of rheumatic fever immediately precedes the chorea. One patient had no chorea during the first pregnancy; in the interval between the first and the second she had an attack of rheumatic fever, and during the second she suffered from chorea (Mosler). But in the majority there is no predisposing influence, except the pregnancy, to which the disease can be ascribed. Fright, or some other distressing emotion, is the apparent exciting cause of the disease in about the same proportion in gestational chorea as in the chorea of earlier life. If the disease has occurred during one pregnancy it may or may not occur during one or more succeeding pregnancies. It scarcely ever occurs for the first time over twenty-five years of age. The number (28) of cases occurring at different ages is as follows: 17, three; 18, three; 19, three; 20, eight; 21, two; 22, two; 23, six; 24, one.*

In the recorded cases of distinct chorea occurring during pregnancy after twenty-four years of age, the patients have suffered from chorea during a previous pregnancy. The disease is most common during the first pregnancy, and is very rare after the second except as a recurrence. Of thirty-eight recorded attacks, twenty-five were during the first, and ten were during the second pregnancy, the first having been free. In two attacks in the third, and one during the fourth pregnancy, the patients had previously suffered from chorea during a similar condition. In the only recorded case† in which a patient is said to have suffered for the first time during the fourth pregnancy, it is doubtful whether the disease was really chorea, since the movements were confined to the legs.

The chorea may commence at any period of pregnancy, from the beginning until near the close, but it begins more frequently in the third than in any other month, and very rarely in the ninth month. Of thirty-six attacks‡ the numbers commencing in each month were—1st, four; 2nd, three; 3rd, nine; 4th, five; 5th, four; 6th, four; 7th, three; 8th, three; 9th, one. Thus two thirds of the attacks commence before the end of the fifth month, and one quarter of the total number commence in the third month. If chorea recurs during several pregnancies, there is no uniformity in the date of its commencement; for instance, in one remarkable example of recurrence, the patient (who had had rheumatic fever, followed by chorea at sixteen) became choreic in the fourth month of her first pregnancy (at twenty-two), in the third month of the second, at the commencement of the third, and in the fourth month of the fourth pregnancy (Lawson Tait). The cases apparently excited by fright show no difference in the date of their commencement, but a rather larger proportion do not exceed twenty years of age than of the cases in which no exciting cause can be traced. In one instance albuminuria existed, left (with a cardiac murmur) by an attack of scarlet fever in the fourth month of pregnancy; the chorea commenced in the eighth month (Woodman).

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* The figures are based on an analysis of recorded and original cases; most of the former have been collected by Barnes ('Obstetrical Trans.,' 1869), but some of his cases have not been included, either because insufficiently reported, or because their nature is open to question.

† Levick, 'Am. Journal of Med. Science,' 1862.

‡ In the statement in the text those cases which have been recorded as commencing when the pregnancy had existed for a certain number of months are considered as commencing in the subsequent "month" of gestation.

In extremely rare cases, chorea (of ordinary form) has been observed to commence after delivery or after abortion, and the subjects of this post-puerperal chorea have for the most part been beyond the age to which the chorea of gestation is chiefly limited.

SYMPTOMS.—The characteristic symptoms of chorea, the spontaneous movement, inco-ordination, and muscular weakness, are all present in severe cases, and are often associated with some mental failure. In slight cases the symptoms may be combined in various degrees.

The first thing to attract attention is sometimes the spontaneous twitching, sometimes the interference with voluntary action. The spontaneous movements are first noticed in the hands or face, rarely in the legs, and so closely resemble those involuntary actions which "nervous" children present under emotion that they are often regarded as such. The interference with movement is rarely at first a conspicuous inco-ordination; a sudden purposed movement is more considerable than was intended, or a persistent action is suddenly disturbed by an unwilled movement. The insubordination of the motor centres is seen also in the occasional unintended relaxation of muscles, or in a delay of intended relaxation. Thus objects which are being carried are suddenly dropped, and in one lad the first indication of the commencing disease was that in throwing a cricket ball the fingers relaxed their grasp too late, and the ball simply fell to the ground. The spontaneous movements may at first be recognised only on close observation, but as the disease advances they become conspicuous enough. They are quick and irregular, sometimes complex in character, and each movement is brief in duration. The mouth is drawn to one side with an associated movement of the lips, the eyes are closed for a moment. The fingers are irregularly extended, the hand is pronated or supinated, or the whole arm is suddenly extended or rotated, and these movements are often combined. In the legs the movements are usually more simple, momentary muscular contractions; they may cause merely a slight jerk of the body, but, if considerable, walking and even standing may be seriously interfered with.

The spasmodic movements are always irregular in time as well as in character and degree. At first they are occasional only, but their frequency increases with their severity, until at last they may be so continuous and violent that the limbs are in constant movement. The spasm in the muscles of the neck may cause frequent movement of the head to one side, and the eyes may move with the head. In many cases the spasm of the eyeball muscles is not quite equal, and momentary diplopia results. The muscles of the trunk may also be involved. When their affection is slight, the patient from time to time sways to one side while sitting or standing; when considerable, standing or sitting is impossible, and the patient may even be thrown out of bed by some violent contortion. The limbs may be thrown about with such force that serious bruises result from their contact with adjacent objects, and in one recorded case the choreic spasm of the muscles of the jaw was so violent that several of the teeth were broken (Tuckwell). The movements are always increased by excitement, and by attempts at voluntary movement, and are lessened by repose, physical and mental. They almost invariably cease during sleep, natural or nduced, but their severity may prevent sleep for days together.

Voluntary movements are executed rapidly and in a spasmodic manner. This character seems to be in part impressed upon them by the tendency to spasm, in part to be the result of an attempt to escape the disturbing influence by rapidity of movement. Thus the tongue is protruded suddenly and jerked back again, or the hand makes a dash at any object which it attempts to grasp. Usually, and always in severe cases, the resulting movement is irregular, partly from disturbing spasm, partly from an inco-ordination which is not always related in degree to the amount of spontaneous movement. In some ases the latter may be slight while inco-ordination is great, and, onversely, voluntary movement may be almost steady although there s much spontaneous spasm. Sustained muscular action is often mpossible; an object may be grasped with firmness, but first one inger and then another relaxes in spite of the will.

The muscles of respiration, especially the diaphragm, frequently hare the irregularity observed in the action of the more strictly voluntary muscles; the respirations are unequal; deep and shallow breathing alternates, or deep breaths are separated by pauses; thoracic and abdominal breathing may alternate irregularly. The heart's action is sometimes irregular, but this is almost always produced by, and distinctly consequent on, the inequality of the respiratory movements.

Besides the interference with movement occasioned by spasm and inco-ordination, there is usually distinct deficiency of muscular power. The degree of loss of power may not be proportioned to the spasm; it may be considerable when the latter is barely recognisable. This is frequently noticeable at the onset, but sometimes there is much weakness and scarcely any spasm, a form which I have proposed to call "paralytic chorea."* There is never, however, anything like complete loss of power, and often the loss of use of a limb is out of proportion to the actual muscular weakness.

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These motor symptoms may be from the first general, and throughout may involve both sides equally. The arms are almost always affected earlier and in greater degree than the legs, and the movements in the arms are wider in range and more irregular in character. The difference that exists between the physiological movements of the upper and the lower limbs is thus reproduced in the spasm. In at least half the cases, however, the affection of the two sides is not equal. It may be throughout limited to one side (hemichorea), or it may affect one side first, and afterwards the other. In the latter cases it may continue on the side first affected when the other is involved, unilateral chorea thus becoming general; or it may cease on the first, when it passes to the other. The cases in which it is limited to one side present considerable variety in the area affected: the movements may involve the arm only, the leg escaping; they may affect the arm and leg on the same side, or one arm and both legs, the other arm remaining free and the leg on the side of the unaffected arm being usually less involved than the other. These variations in the distribution of partial chorea correspond closely to the distribution of partial convulsive seizures. There seems to be no difference in the frequency with which the right or the left side is the seat of hemichorea, or is affected most or first in general chorea. No relation can be traced between the presence of heart disease, or the influence of fright, in causing the disease, and the side affected. Of sixty-four cases I find the total numbers nearly equal for each side:

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* 'Brit. Med. Journal,' April 23rd, 1881.

	Right.	Left.
One side only	11	13
One side first	10	10
One side most	10	10
	31	33 = 64.

The *electric irritability* of the muscles and nerves can only be thoroughly studied in cases of hemichorea in which the unaffected limbs are available for comparison. Sometimes no alteration of irritability can be discovered. But in other cases a distinct increase of irritability may be found in nerve and muscle to both faradism and voltaism,* the difference amounting to one or two centimetres of the secondary coil of a faradic apparatus, or two to four cells of a voltaic battery. I have observed this increase of irritability to be absent at the commencement, to come on during the course of the disease, and to subside with recovery. A "qualitative" change in the mode of response to voltaism has also been described, instead of the contraction on closure of the circuit, occurring at the negative pole with a weaker current than at the positive, the latter may occur as readily as the former (*i. e.* instead of 1 K.C.C., 2 A.C.C., we have K.C.C. = A.C.C.).

Speech is often impaired in chorea. The movements of the muscles of articulation are disturbed just as those of other voluntary muscles.

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* I have observed this in several cases, and it has also been noted by Benedikt, Rosenthal, and Schmitt.

Irregularity of movement of the vocal cords has been observed with the laryngoscope (v. Ziemssen), while the disturbance of respiration further deranges utterance. Words are uttered quickly, just as voluntary movements are performed quickly; speech is interrupted by sudden, deep inspirations, which often cut off the last syllables of a word. Sometimes the words are jerked out in separated parts. Actual stammering is rare, but I have met with one case in which it preceded the other symptoms. In severe cases the interference with articulation may be very great; the spasm in the muscles of the mouth and tongue on any attempt at articulation may render it impossible for weeks for the patient to utter a single word. There is often a disinclination to speak (due to the conscious difficulty, and partly perhaps to the mental state presently to be described) in addition to the actual interference with articulation.

Sensibility is, as a rule, unaffected. In extremely rare cases, unilateral diminution or increase of sensibility, sometimes involving the special senses, has been observed. Such disturbance is probably allied to that met with in hysteria, and is certainly no part of the ordinary symptoms of chorea. Tender points along the spine or along the course of the nerves (especially where these emerge from deeper structures) have rarely been observed (Cartier), and may have been due to a slight degree of the multiple neuritis which has been met with, in severe form, as a complication.

Chorea is, as a rule, a painless disease. The muscular spasm may cause fatigue, but occasions no sensation of pain. I have met with only two cases in which there was pain in the limbs at the onset. One patient, a girl, complained of pain in the left hand, which gradually, in the course of a few hours, passed up the arm to the side of the head, and three days after was felt in the leg. The choreic movements commenced in each limb at the same time as the pain. The disease afterwards became general without any pain in the other side. There was no affection of the heart. In the other case, also a girl, intermittent neuralgic pains in face, arm, and leg occurred for some weeks before the onset of right-sided chorea. The latter was accompanied by some hysterical hemianæsthesia.

The pupils are often large, but it is doubtful whether this character is connected with the disease. Once I observed inequality of the pupils, the larger being on the side opposite to the limbs most affected.

The mental state, in slight cases of chorea, may be normal, but in severe cases there is usually some irritability and often distinct mental dulness. The degree of change does not bear any relation to the severity of the other symptoms. The mental dulness is usually conspicuous in the patient's aspect, and may amount to practical dementia, so that stools and urine may be passed into the bed, not from paralysis of the sphincters, but from mental apathy. In other cases the irritability may pass into excitement, the mind seems to share the disturbance so conspicuous in the muscles, and there is excited delirium, which, in its restlessness and delusions, may resemble mania, and in some cases must be regarded as such. In some of these cases the physical symptoms are trifling compared with the mental disturbance.

Symptoms outside the Nervous System.—The temperature in slight cases is normal throughout, but in severe forms it may be raised one or two degrees. Even in these it rarely reaches, and still more rarely exceeds, 102° F. I have once observed occasional elevation of temperature from time to time, during a prolonged attack, without any complications or endocarditis to which it could be referred.*. Hyperpyrexia occurs in rare cases, probably as a rheumatic complication in most instances, but in uncomplicated acute chorea a rise to 105° has been observed before death. The patients are often previously anæmic, or they become pale and lose weight rapidly during the course of the disease, especially if this is so severe as to interfere with sleep.

Heart.—The cardiac symptoms are of great importance. The pulse, as already stated, may be irregular in consequence of the irregularity of the breathing. It is usually increased in frequency. I have several times noted that the effect of posture on the pulse is less than in health; the pulse-rate may be nearly or quite the same in the upright and the recumbent postures. In some cases the sounds and impulse of the heart are normal. More frequently there is some abnormality. (1) There may be murmurs, due to the anæmia, in the pulmonary artery and aorta, often associated with a venous hum in the jugular vein. Sometimes the systolic murmur at the base is continuous with one heard over both ventricles, having its maximum at the inner end of the fourth intercostal space, heard up to but not beyond the left apex, and probably produced in the ventricles, in consequence of the blood-state. With this there may be such a change in the character of the impulse as indicates slight dilatation of the heart consequent on the anæmia. (2) There may be a faint systolic murmur at the apex of the heart, heard with some beats and not with others; this is commonly said to be due to irregular contraction of the papillary muscles, which sometimes fail to compensate for the shortening of the ventricle, and thus permit occasional regurgitation —a mechanism that can neither be proved nor disproved. (3) In a considerable number of cases there is distinct evidence of an organic valvular lesion. The frequency of organic disease is less in childhood than in youth,† and hence has been variously estimated by different observers, according to the class of cases which have come under their notice.

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* In the 'Collect. Invest.' cases, pyrexia was noted in only 12 per cent., but it is probable that this only represents the frequency of such considerable and prolonged pyrexia as would attract attention.

† This statement is at variance with the opinion of others, by whom, for the most part, "heart symptoms" of hæmic, dynamic, and organic origin have not been properly distinguished.

In some cases with organic disease there is good reason to believe that the heart disease preceded the chorea (see *ante*, p. 595); in other cases the true relation between the two is doubtful; in other cases again the organic murmur may be observed to develop during the course of the disease—an indication of endocarditis which, as will be seen, pathology amply confirms; in nine out of every ten fatal cases the cardiac valves are diseased. Hence it is of great importance that the heart should be examined repeatedly during the course of the disease, and also after its termination, but it must be remembered that endocarditis may exist when no murmur can be heard. Preceding valvular disease is most frequently mitral regurgitation; occasionally there is mitral constriction alone or combined with regurgitation. Aortic disease is much less common; I have only met with two instances of aortic regurgitation among about 250 cases of chorea that have come under my observation. In one case there was an aortic obstructive murmur, musical in tone, combined with mitral regurgitation.* In the cases in which the relation of the disease to the chorea is doubtful, or in which the heart is affected during the chorea, the lesion is almost always mitral regurgitation. The frequency of a hæmic murmur at the aortic orifice renders it almost impossible to recognise the development of an organic obstructive murmur during chorea, but its occurrence was probable in a case in which I found, at a second attack of chorea, unquestionable evidence of aortic obstruction which was absent in the first attack, the patient having had in the interval no rheumatic fever. Post mortem, endocarditis has been found at the aortic as well as at the mitral orifice. A murmur due to mitral regurgitation usually persists after the chorea has ceased, but may afterwards pass away, sometimes to be succeeded by evidence of obstruction as the altered orifice contracts. In a prolonged case, however, I have known a murmur, certainly mitral, to cease before the movements.

The *urine* in chorea contains an excess of urea and of phosphates (Walshe, Handfield Jones, and others) bearing a general proportion to the severity of the attack. Albumen is present only from independent kidney disease or renal embolism. The peculiar pigment "urohæmatoporphyrin," discovered by McMunn in cases of rheumatism, has been found also in chorea by A. E. Garrod, who has pointed out that it is additional evidence of the connection between the two maladies, since it is absent in other nervous diseases.

Complications.—Endocarditis is so common in chorea that it can scarcely be regarded as a complication. It is usually of a benign form, and causes no subjective symptoms, although it may lead to permanent valvular disease. Occasionally its existence is emphasised by the occurrence of embolism. I have seen hemiplegia from embolic softening of the brain occur during the course of chorea; a similar consequence of endocarditis distinctly originating during an attack of chorea has been recorded by others. Embolism of the central artery of the retina has also occurred during an attack.* It is exceedingly rare for the endocarditis to assume the more malignant type of the "ulcerative" form, and the condition is met with only after childhood, in the subjects of preceding valvular disease. In the only instance of this dangerous complication that has come under my notice the patient was also suffering from Bright's disease, but died from the septic embolic processes.

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* In the 'Brit. Med. Ass. Collect. Inv.' there were 116 cases of pure mitral disease and only six of pure aortic disease. Unfortunately the facts regarding heart disease and chorea are given in so condensed a form in the Report that it is difficult to draw any general conclusion from them. Distinct organic disease, either preceding or secondary, was found in 32 per cent. of the cases. But the difficulty of deciding whether there is or is not organic disease is often very great, and the large number of observers entails considerable uncertainty as to the value of the statistics on this point.

Acute articular rheumatism may not only immediately precede chorea, but may come on during the course of the disease without any distinct exciting cause. It is usually attended with moderate elevation of temperature, is trifling in severity, and brief in duration. Evidence of fresh endocarditis may sometimes be detected during the course of the rheumatism. In some patients there may be found the small subcutaneous nodules, varying in size from that of a pin's head to that of a pea, met with also in those who are liable to rheumatism.† They may be felt not only beneath the skin, especially of the arms, but also on the tendons, especially of the flexors of the fingers, and the peroneal muscles of the hands. They have been observed to come on with the chorea and pass away with it.‡

In most cases of chorea the ophthalmoscopic appearances are those of health. In a few there is optic neuritis, usually slight in degree, just enough to be unequivocal. It passes away when the chorea is over. In only one case have I seen considerable neuritis, comparable to that seen in a case of tumour; the inflammation passed entirely away with the chorea. It is probable that the neuritis is related to the cause of the chorea rather than directly to the morbid process in the brain. Many of the patients had considerable hypermetropia, and it is known that this condition disposes to slight neuritic changes in the discs, and may aid other influences in leading to the change; it cannot be the sole cause, because the discs resume their normal aspect without any treatment of the hypermetropia. The rare ulcerative endocarditis may be accompanied by the characteristic minute retinal hæmorrhages due to micrococcal embolism.

Convulsive attacks of any form are more rare than might have been anticipated from the spasmodic nature of the affection. In one case, in which the choreic movements were most severe, occasional attacks occurred in which there were apparent loss of consciousness and peculiar convulsions, partly choreic in form. In one, for instance, the head turned to the right, the right arm and leg presented violent choreic movements, while the left was stretched out, the fingers extended, the thumb inverted, and the whole limb in a state of cataleptic rigidity, so that it remained in whatever position it was placed. The attack lasted two minutes. In another attack there was some arching of the back and tonic flexor spasm in the arms, followed by violent choreic movement in the right arm. Such attacks occurred daily for three weeks.* Epileptiform convulsions are very rare. In one case of chorea in a boy of twelve, greater on the right side, four convulsive attacks, limited to this side, occurred during the fortnight after the cessation of the chorea, and similar attacks afterwards persisted as chronic epilepsy. There was no cardiac murmur. I have met with a few other cases in which epilepsy dated from an attack of chorea.

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* Sym, 'Edin. Med. Journal,' 1888.

† They were first observed by Meynet ('Lyons Médical,' 1875, No. 49). The most important articles on the subject are by Barlow and Warner, 'Trans. International Med. Congress,' 1881, vol. iv, p. 116, and by Hirschsprung, 'Jahrb. für Kinderheilk.,' March, 1881.

‡ Sheele, 'Deut. med. Wochenschrift,' 1885, No. 41, who observed that the development of the nodules was accompanied by transient contracture of the flexors.

Persistent spasm is also occasionally met with. In a young child there was continuous spasm in the arm during the attack, which was one of true chorea. The elbow- and wrist-joints were flexed, and the aspect of the limb was similar to that seen after infantile hemiplegia. The choreic movements were present in the arm, although in much less degree than in the other limbs. As the child recovered the persistent spasm lessened, and the movements in the limb became greater. Similar spasm had been present in a previous attack. Transient rigidity of the neck was present in one recorded case, and in another there was, for a time, rigidity of the legs, with foot-clonus.†

The cases in which the mental disturbance is very great have been termed *maniacal chorea*, or *chorea insaniens*. The form occurs chiefly in females, at or soon after puberty, or during pregnancy. The mental disturbance may not come on until the disease is well developed, or it may precede the chorea. Thus either the mania or the chorea may be primary. In the latter, the movements may be severe or slight; they may even cease while the mental disturbance continues in intense form, or both may continue. If the attack of chorea is acute and severe, there is often considerable elevation of temperature, and much danger to life. In less severe cases there are often delusions, and sometimes wild violent excitement, but rarely the intense garrulity so common in ordinary mania. The excitement usually subsides in the course of a week or two, and leaves dulness and apathy, with a depressed listless look, disinclination to speak, and sometimes persistent hallucinations. Food has often to be administered by the rectum or by force, and stools and urine are commonly passed unnoticed. This condition usually slowly passes away, but occasionally persists for weeks or even for months after the chorea has ceased, and it may even be permanent. In one case, recorded by Golgi, there was no improvement, but slowly increasing mental failure, until death ten years later. A similar sequel has been observed to maniacal chorea occurring during pregnancy.

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* I have given fuller details of these attacks in 'Epilepsy, &c.,' 1881, p. 181. Similar paroxysms of choreiform spasm occurred in a case described by Mitchell and Barr ('Journ. of Nerv. and Ment. Dis.,' 1891).

† Mitchell and Barr.

The cases in which loss of use of a limb is the only conspicuous symptom, "*paralytic chorea*," occur only in childhood, and affect chiefly the arms, one always more than the other. Slight twitchings may occur at the onset and cease, the weakness remaining. More commonly the loss of power is the first symptom, and it may persist or even lessen as the movements develop. Rarely there is considerable weakness of the legs for a time.

Duration.—Few diseases are so variable in duration as chorea. It usually lasts from six weeks to six months. It rarely falls short of the earlier limit, but I have known the disease in one or two instances to last only three weeks. On the other hand, the limit of six months is frequently exceeded. The average duration has been found by more than one observer to be ten weeks.* This is, however, true only of cases which are admitted into hospitals before they have lasted for a considerable time. At the National Hospital for the Paralysed and Epileptic, to which choreic patients are often brought as a last resort, I have met with no less than fifteen cases in which the duration exceeded six months, and seven in which it exceeded a year. Occasionally a slight degree of chorea persists for a much longer time, and I have seen cases in which it has continued for two, three, four, and even six years, without any complete intermission. In most of these cases of very long duration, however, there are occasional remissions of the disease, so that the prolonged course is rather a series of relapses, with imperfect recovery in the intervals. In extremely rare instances the disease never ceases. A patient was attacked with chorea in youth, and the disease continued until death at the age of sixty-six.† A girl was attacked with chorea at the age of nine, and the movements continued and were still present at the age of fifty.‡ As a rule, the more severe an attack, the longer is its duration, but the influences which determine either severity or duration are very obscure. I have been unable, on a comparison of the details of cases, to discover any relation between duration and age, sex, state of heart, preceding rheumatic fever, or exciting cause.

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* From the tables given in the 'Coll. Invest. Report' I have calculated the following percentages of the duration of 396 cases:—Not exceeding one month, 18 per cent.; between one and three months, 57 per cent.; over three months, 25 per cent.; only 6 per cent. exceeded three months. The percentage is nearly the same for each sex. (The calculation is made on the assumption that the cases in which the duration is [for instance] three months are given in the column headed "under three months," which is probable from the way the table runs, although inconsistent with its wording.)

† Meldner, 'Wochenbl. der Gesellsch. der wiener Aerzte,' 1869, No. 19.

‡ Macdougal, 'Lancet,' 1885, No. 17.

Recurrence.—Chorea is a disease prone to recur. In how many cases it again attacks an individual can only be approximately determined, since the subsequent history of many patients is unknown. In the series of original cases that I have analysed, one third suffered from more than one attack. Fifteen patients had more than two attacks, viz. nine, three attacks; one, four; three, five; one, six; and one no less than nine separate attacks. Females are more liable to relapse than males, just as they are more prone to chorea; the proportion of the sexes in second attacks, however, is nearly the same as in first attacks, but in the cases in which more than two occur the predisposing influence of sex is very strong, the proportion of males being much below the average, while almost all patients who have more than three attacks are of the female sex. The disease very seldom recurs between eighteen and thirty, except in connection with pregnancy.

Preceding rheumatic fever seems to have no influence on recurrence. I have found a history of it in precisely the same proportion of the cases that recurred as in the whole series. Organic heart disease is unquestionably more frequently present in recurrences than in first attacks. It was distinct in one half of the cases in which the attack was the second or the third, and in all the cases in which the number of attacks exceeded three. This might, indeed, be expected from the fact that endocarditis often occurs in chorea without other discoverable cause; the more attacks of chorea a patient has had, the greater will be the probability of consecutive heart disease. The same conclusion, that the organic heart disease is, for the most part, the result of the repeated attacks, is also shown by the fact, mentioned above, that a history of rheumatic fever is not more frequent in recurrences, and also by the not infrequent cases in which, during a first attack, there is no distinct evidence of valvular disease, but in which, at the second attack, such disease is unmistakable, although there has been no rheumatic fever in the interval.

The interval between the termination of the first and commencement of the second attack of chorea varies from a few weeks to two or three years. It is rarely less than two months or more than two years. The average interval is about one year. In only one third of my own cases was the interval more than a year, and in only two did it exceed a year and a half. If, therefore, a patient has remained well for eighteen months after the chorea is over, it is improbable that another attack will occur. The intervals between subsequent recurrences present similar variations. In one case, however, a third attack commenced only two weeks after the termination of the second. The intervals usually vary much in the same patient, and, as a rule, there is no uniformity in the time of year at which the recurrence occurs. Remarkable exceptions, however, are occasionally met with, which are of considerable interest in connection with the relation of chorea to season. One girl first suffered from chorea at nine years old. The attack commenced in October, and lasted four months. Every autumn, for the next six years, she became choreic, and remained so until the middle or end of winter. Her eighth attack, however, began in May, and in it (at the age of seventeen) she came under my care, and presented the characteristic signs of mitral constriction, although she had never had rheumatism: the attack lasted four months. The following year a ninth attack commenced in June; it was the shortest of all, lasting only a month, and has, I believe, been followed by no recurrence. Another girl was attacked with chorea at seven, after distress at the death of her mother, and in each of the next five years, chorea came on in late spring or early summer, and lasted for three or four months. I saw the sixth attack, which commenced in May. There was a loud mitral regurgitant murmur, and she also had never had rheumatic fever.

Fright is to be traced as a cause of recurrence in at least as large a proportion as of first attacks. One girl, for instance, had attacks at 15, 17, 17½, and 18, all of which were apparently excited by fright. The attack at 17½ commenced the morning after she had been frightened by a severe thunderstorm; the last attack began a week after she had been much startled by the touch of an unseen person when she thought she was alone. On the other hand, in the case in which there were nine attacks related to season, not one was excited by fright. In the girl who had six attacks, the first five occurred without an exciting cause; the sixth commenced a few hours after a sudden alarm.

The course of the affection in successive recurrences, and the side affected first or chiefly, present no uniformity. In exceptional cases the commencement is on the same side in every attack. Thus, in one patient four successive attacks began, and were more severe, on the left side. In the patient who had nine attacks, the first seven, which occurred in winter, affected the right side chiefly, but in the eighth and ninth attacks, which occurred in summer, the left side suffered most. The symptoms of the recurrent disease are similar to those of first attacks, but its degree is often slighter and its duration shorter. In this respect, as in most others, chorea shows its characteristic variability; relapses are occasionally most severe. A lad had attacks of chorea at five and seven, each lasting three months; and at ten, without exciting cause, a third attack came on, of great severity, which lasted, varying in intensity, for more than twelve months, in spite of most careful treatment. In this case there was no organic heart disease, but I have more than once observed distinct signs of endocarditis to come on during a recurrence, at the onset of which the heart was healthy.

Termination and Sequelæ.—The vast majority of the cases terminate in recovery. The movements gradually lessen in severity, until at last they are to be observed only in moments of emotional excitement, and finally they disappear. Usually inco-ordination ceases, and any weakness passes away, before the spontaneous spasm has entirely vanished.

The final disappearance is often extremely gradual, and slight occasional movements may be discerned, if carefully looked for, long after the patient is practically well.

In very rare cases considerable muscular weakness, general or local, may succeed chorea. It is a post-choreic paralysis. The loss of power is sometimes very great. There is no change in muscular irritability, but there is, in some cases, impairment of sensibility. It is perhaps analogous to the mental weakness which sometimes succeeds chorea, and, like it, always passes away in the course of a few weeks.

Occasionally the disease leaves behind it, for a time, a liability to sudden starts of the limbs, similar to those which many persons experience on going to sleep. In one child they were troublesome for many months. A sudden start in the legs would throw her down, and once the start was such as to make her jump into an open cellar, at the edge of which she was standing. The fact that epilepsy is a rare sequel to chorea has been already mentioned.

When the chorea is attended by considerable mental disturbance, this may continue, as dulness or delusions, for some weeks after the motor phenomena are at an end. Rarely the mental derangement persists as a chronic affection for months or years, but it gradually passes away.

We have seen already that juvenile chorea, in extremely rare cases, instead of ceasing, persists, in more or less severity, as a chronic disease, in spite of all treatment. In the case mentioned, in which the chorea commenced immediately after the discharge of a pistol near the ear, the disease had lasted for three years when the patient came under treatment, and it was still present when he passed from observation six months later. Adults are sometimes seen in the streets presenting all the characteristics of severe chorea, but following their avocations in a way which shows that the condition is one to which they have become habituated. This persistent form, while closely resembling ordinary chorea, does not prevent locomotion. Most of the subjects have been males. It has been pointed out that the proportion of males is unusually large in cases of prolonged duration.

The chorea of childhood rarely terminates in death. The average mortality of a disease so rarely fatal is not easy to ascertain, but it is certainly less than 3 per cent.,* and most of the fatal cases occur at the time of puberty. The proportion of deaths in the two sexes at this time corresponds nearly with their liability to chorea (Sturges), but in earlier childhood the mortality, compared with the number of cases, is relatively lower in boys than in girls. The immediate cause of death is sometimes exhaustion from the severity of the disease and the loss of sleep, sometimes associated rheumatism or rheumatic hyperpyrexia, sometimes intercurrent maladies, as pyæmia from local injuries. Most of the fatal cases are first attacks; death in recurrent chorea is very rare. The chorea of pregnancy is much more fatal, the mortality being 20 or 25 per cent. But even in these cases death seldom results directly from the chorea; it has usually been due to the effects of delivery or abortion, in the cachectic state of the system.

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* Nine deaths occurred among the 439 cases of the B. M. A. Coll. Inv.,—a mortality of 2 per cent.

VARIETIES.—*Hysterical Chorea.*—Hysterical patients occasionally suffer from general spasmodic movements, which sometimes resemble those of true chorea, but more often differ from it in the greater regularity of the muscular contractions. The mere existence of symptoms of hysteria does not prove that the chorea is not of the ordinary form, if it corresponds to this in its features; a patient suffering from hysteria may also suffer from true chorea, and, in a predisposed person, hysterical disturbance may develop during an attack of true chorea, and may even be evoked by it. The only circumstance that would justify the diagnosis of the hysterical nature of such an attack would be its origin by imitation, by witnessing the symptoms of chorea in another patient. In ordinary hysterical chorea the movements present certain distinguishing features. They are generally more sudden and shock-like, resembling those of the so-called "electrical chorea." This is a rare feature of true chorea, in which such sudden contractions of many muscles are met with rather as a spasmodic sequel than as part of the symptoms. The second feature of the hysterical form, more frequent and of still greater significance, is the rhythmical character of the spasm. The movements are more or less regular, instead of presenting the characteristic irregularity of ordinary chorea. The rhythmical character is especially seen in the movement of the hands, and even of the fingers. The spasm is usually moderate in degree, never so intense as in the severer forms of the genuine malady; nevertheless the hysterical form is often a very obstinate malady. It may last for many months; indeed, its average duration is considerably greater than that of the ordinary form. It should be noted that the term "hysterical chorea" is sometimes applied to other forms of hysterical spasm which bear little resemblance to true chorea; these are described in the chapter on Hysteria.

The relation which this form bears to true chorea is sometimes uncertain, and the occurrence of the two under the same conditions of age may occasionally render the diagnosis difficult. It is not so, however, in the case of the other important varieties of chorea, the chronic forms of middle and late life, *hereditary chorea*, and *senile chorea*. In spite of a superficial resemblance, these present such important differences from the common chorea of childhood as to make it convenient to describe them separately. This is also the case with the peculiar affection termed "*electrical chorea.*" The name has also been applied to maladies that have no real relation to chorea. Choreoid movements, on one side, may be the result of an organic lesion, transient, before the onset of hemiplegia, *pre-hemiplegic chorea*, or persistent, as a late sequel of the paralysis and a variety of "athetosis," *post-hemiplegic chorea*. It is only necessary here to mention this application of the word. Lastly, it has been employed as a designation for the movements that are better termed *habit-spasm* (*q. v.*). One form of this, manifested by a peculiar rough or "barking" cough, has been especially called "*laryngeal chorea*;" without, however, any justification.

PATHOLOGICAL ANATOMY.—As a rule, the nerve-centres in chorea present no abnormal appearances on naked-eye examination, and a careful microscopical investigation often yields only a negative result. In some cases, alterations have been found, conspicuous or microscopical, the significance of which will be presently considered. They are chiefly of two classes: (1) changes in the vessels, with or without secondary effects upon the brain, the result either of a morbid state of the blood or of local derangement of function; (2) alterations in the nerve-elements themselves, which are apparently primary, and the effect of their abnormal activity. In severe cases, of very acute course, general hyperæmia of the brain has been observed, or dilatation of small vessels has been seen with the microscope, sometimes in the cortex, especially of the motor region, once in the white substance with degeneration of the walls (Dana), and sometimes with a perivascular accumulation of leucocytes. Small arteries have been found occluded by a process of embolism or thrombosis, varying in different cases. The plugging has been attended by minute hæmorrhage in some cases, and very rarely a large hæmorrhage has been found, connected, no doubt, with endocarditis (see p. 603). Minute foci of hæmorrhage or of softening are met with in very acute cases, with endocarditis, especially of the "ulcerative" form; and in this I have also seen small round hæmorrhages scattered over the pia mater, like those seen in the retina. Micrococcal embolism of small arteries is probably one mechanism by which these lesions are produced, and occasionally endocarditis has caused embolism of a larger artery, with consequent focal softening, corresponding to the hemiplegia mentioned as an occasional complication. In one case (Gray) the basilar, vertebrals, and middle cerebrals were all thus closed. The plugs in minute vessels have been probably thrombotic in some cases. They are not invariable, for they have often been carefully looked for without success. In some acute cases, especially with delirium, the peculiar round hyaline bodies, that are met with in the arterioles and capillaries of the brain in acute infectious diseases, have been found in the perivascular sheaths.* Effusion into the cerebral ventricles has been very rarely observed, hyperpyrexia, sometimes intercurrent maladies, as pyæmia from local injuries. Most of the fatal cases are first attacks; death in recurrent chorea is very rare. The chorea of pregnancy is much more fatal, the mortality being 20 or 25 per cent. But even in these cases death seldom results directly from the chorea; it has usually been due to the effects of delivery or abortion, in the cachectic state of the system.

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* Flechsig, Wallenberg, Jakomenko. They were first described by v. Reckling-

VARIETIES.—*Hysterical Chorea.*—Hysterical patients occasionally suffer from general spasmodic movements, which sometimes resemble those of true chorea, but more often differ from it in the greater regularity of the muscular contractions. The mere existence of symptoms of hysteria does not prove that the chorea is not of the ordinary form, if it corresponds to this in its features; a patient suffering from hysteria may also suffer from true chorea, and, in a predisposed person, hysterical disturbance may develop during an attack of true chorea, and may even be evoked by it. The only circumstance that would justify the diagnosis of the hysterical nature of such an attack would be its origin by imitation, by witnessing the symptoms of chorea in another patient. In ordinary hysterical chorea the movements present certain distinguishing features. They are generally more sudden and shock-like, resembling those of the so-called "electrical chorea." This is a rare feature of true chorea, in which such sudden contractions of many muscles are met with rather as a spasmodic sequel than as part of the symptoms. The second feature of the hysterical form, more frequent and of still greater significance, is the rhythmical character of the spasm. The movements are more or less regular, instead of presenting the characteristic irregularity of ordinary chorea. The rhythmical character is especially seen in the movement of the hands, and even of the fingers. The spasm is usually moderate in degree, never so intense as in the severer forms of the genuine malady; nevertheless the hysterical form is often a very obstinate malady. It may last for many months; indeed, its average duration is considerably greater than that of the ordinary form. It should be noted that the term "hysterical chorea" is sometimes applied to other forms of hysterical spasm which bear little resemblance to true chorea; these are described in the chapter on Hysteria.

The relation which this form bears to true chorea is sometimes uncertain, and the occurrence of the two under the same conditions of age may occasionally render the diagnosis difficult. It is not so, however, in the case of the other important varieties of chorea, the chronic forms of middle and late life, *hereditary chorea*, and *senile chorea*. In spite of a superficial resemblance, these present such important differences from the common chorea of childhood as to make it convenient to describe them separately. This is also the case with the peculiar affection termed "*electrical chorea.*" The name has also been applied to maladies that have no real relation to chorea. Choreoid movements, on one side, may be the result of an organic lesion, transient, before the onset of hemiplegia, *pre-hemiplegic chorea*, or persistent, as a late sequel of the paralysis and a variety of "athetosis," *post-hemiplegic chorea*. It is only necessary here to mention this application of the word. Lastly, it has been employed as a designation for the movements that are better termed *habit-spasm* (*q. v.*). One form of this, manifested by a peculiar rough or "barking" cough, has been especially called "*laryngeal chorea*;" without, however, any justification.

Pathological Anatomy.—As a rule, the nerve-centres in chorea present no abnormal appearances on naked-eye examination, and a careful microscopical investigation often yields only a negative result. In some cases, alterations have been found, conspicuous or microscopical, the significance of which will be presently considered. They are chiefly of two classes: (1) changes in the vessels, with or without secondary effects upon the brain, the result either of a morbid state of the blood or of local derangement of function; (2) alterations in the nerve-elements themselves, which are apparently primary, and the effect of their abnormal activity. In severe cases, of very acute course, general hyperæmia of the brain has been observed, or dilatation of small vessels has been seen with the microscope, sometimes in the cortex, especially of the motor region, once in the white substance with degeneration of the walls (Dana), and sometimes with a perivascular accumulation of leucocytes. Small arteries have been found occluded by a process of embolism or thrombosis, varying in different cases. The plugging has been attended by minute hæmorrhage in some cases, and very rarely a large hæmorrhage has been found, connected, no doubt, with endocarditis (see p. 603). Minute foci of hæmorrhage or of softening are met with in very acute cases, with endocarditis, especially of the "ulcerative" form; and in this I have also seen small round hæmorrhages scattered over the pia mater, like those seen in the retina. Micrococcal embolism of small arteries is probably one mechanism by which these lesions are produced, and occasionally endocarditis has caused embolism of a larger artery, with consequent focal softening, corresponding to the hemiplegia mentioned as an occasional complication. In one case (Gray) the basilar, vertebrals, and middle cerebrals were all thus closed. The plugs in minute vessels have been probably thrombotic in some cases. They are not invariable, for they have often been carefully looked for without success. In some acute cases, especially with delirium, the peculiar round hyaline bodies, that are met with in the arterioles and capillaries of the brain in acute infectious diseases, have been found in the perivascular sheaths.[*] Effusion into the cerebral ventricles has been very rarely observed, and it is only in cases of extremely chronic course, which have lasted for years, that any trace of general meningitis has been seen, although œdema of the pia mater over the motor cortex has been seen in an acute case.* Changes in the nerve-cells have been found by some observers, hyaline swelling and degeneration in the central ganglia (Meynert), and in the claustrum and island of Reil (Elischer). The connective tissue of the brain has been found increased and the specific gravity of the central ganglia lowered, but the significance of these changes is doubtful. No special importance can be attached to the fatty degeneration of the perivascular sheaths described by some observers, or to the dilatation of the spaces around vessels, since both these changes are common at all ages.

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[*] Flechsig, Wallenberg, Jakomenko. They were first described by v. Reckling-

In the spinal cord, when changes have been found, they are chiefly such as result from functional over-action, and are met with in acute or very prolonged cases. They consist in general hyperæmia, extravasation of blood around minute vessels, leucocytal aggregations around the walls of the vessels, and fibrinous plugs within them. The nuclei of the interstitial connective tissue have been increased. The large nerve-cells were found by Elischer to present signs of degeneration somewhat questionable in significance (pigmentation and indistinctness of nuclei). Naked-eye changes (beyond congestion) are seldom found, but local softening in the cervical region has been described. Meningitis has been met with as a complication.

In the peripheral nerves Elischer found an increase of connective tissue and slight changes in the axis-cylinders—hyaline swelling, and less ready staining than in health.

Such of the above changes as are not accidental concomitants of chorea are probably the result of the excessive and perverted functional activity of the nerve-elements, which is always attended by vascular dilatation in the part affected, and this may have its own consequence in thrombosis, extravasations, or leucocytal accumulations. Such changes may or may not result, as is illustrated by the pathological anatomy of hydrophobia (*q. v.*), and also of chorea in the dog, an affection which differs from the ordinary form of chorea in man in the shock-like character of the muscular spasm, which resembles that of the so-called "electrical chorea." Of two typical cases examined by Sankey and myself, one presented only slight changes (granular swelling) in the motor nerve-cells of the spinal cord; in the other these changes were more intense, the cells were extensively vacuolated after hardening, and the spinal cord, medulla, and cerebellum presented areas and foci of intense nuclear (leucocytal) infiltration in various parts of the grey or white substance. These differences have been found by others; the cellular infiltration must be secondary, but once produced, developing to independent excess.

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hausen, and a full description of them, as met with in acute diseases, and of the observations by others, is given by Manasse, 'Virchow's Arch.,' Bd. cxx.

* Powell, 'Brit. Med. Journ.,' 1889.

nd a proportionate but secondary derangement of their cur.

function is evidently peculiar. All functional dis- with nutritional change, and this must occur also tructures that are secondarily affected, whether or not it gree that can be recognised by the microscope. It is indi- the increased electric irritability of the nerve-fibres of the s. But it seems never to reach a destructive degree, or to involve ue secondary degeneration. Any destruction of nerve-elements is the result of vascular changes, primary, or secondary to the functional over-action. Moreover this alteration in the nutrition and derangement of function of the nerve-cells tends to pass away when their cause has ceased to act.

We have no evidence regarding the precise part played by the spinal cord in the production of the symptoms, whether it acts merely as a conductor, or whether the spinal cells attain an independent energy under the prolonged excitation, and intensify the spasm, or, from defective power, fail to transmit the voluntary influence. Such co-operation of the spinal elements is, however, only suggested by the rare cases with bilateral spasm or weakness in the legs. The spinal cord takes an independent part in the production of the symptoms of canine chorea, but this malady, as already stated, is essentially different from the chorea of man.

(2) We can give no real explanation of the nature of the change in the function of the nerve-cells whence the symptoms arise. We only restate in pathological terms the observed symptoms when we say that these indicate that there is a spontaneous action of nerve-cells, a tendency for this action to spread unduly among the cells, and a diminution in the possible strength of action. In the language of modern physiology, we may say that the "resistance" is lowered alike in the cells themselves, and in the connections between the cells, by which the character and degree of their associated action is determined. The derangement of function in the motor cells of the cortex (according to the view here adopted) often begins on one side and spreads to the other, just as does the discharge which causes a convulsion; in the latter the over-action of the cells has a tendency to excite the over-action of other cells that are connected with the first, and a similar tendency to the extension of functional action may be the mechanism by which the disorder of movement extends in chorea to structures also predisposed to suffer.

(3) By what morbid influence is the derangement of function of the nerve-cells produced? This is the essential problem of the pathology of the disease, but the changes observed in the fatal cases are too variable and too uncertain in their significance to be trusted alone. We must also consider the facts of causation, and the analogies supplied by allied affections.

The first important etiological fact is the distinctly predisposing cess that causes ordinary chorea. Such movements have been caused by lesions of the cortex, central ganglia, and crus cerebri.*

The motor impulses that excite the muscles pass to the spinal cord from the motor region of the cortex. It is here that movements are arranged, and if they are disarranged, and the disorder proceeds from the brain, we naturally refer it to a disordered action of the cells of the motor cortex. Is their derangement primary? Is this the actual seat of the disease, or is the disorder here merely secondary to a morbid process elsewhere? It is doubtful whether there are at present any facts to justify us in going beyond the motor cortex in our search for the primary disturbance. At the same time we know very little of the mechanisms that guide the cortex in arranging movements. It does not seem that there is any co-ordination of movement in the brain after the motor impulse leaves the convolutions, and any co-ordination in the spinal cord is subsidiary and involuntary. The influences that guide the cortex in arranging movements—the influence of the cerebellum, for instance,—however exerted, must be exerted on the cortex, or on nerve-processes anterior in time to those in the cortex, but we have no evidence of their derangement in chorea.

It was formerly thought that the corpus striatum is the part primarily diseased, but this opinion was based upon the theory that the movements are arranged in the central ganglia after the impulses leave the cortex. We now know that this is impossible, and therefore the ground on which this theory was based has disappeared. Moreover, we know of no anatomical arrangement by which the grey matter of the corpus striatum can influence the cortex. The optic thalamus, however, stands on a different footing. Choreoid movements have been caused by disease of the thalamus, and it is connected with all parts of the brain. It has been conjectured that it is a centre through which peripheral impressions, which do not affect consciousness, influence the action of the cortex. But of its relation to ordinary chorea we have no direct evidence, and the fact that its disease seems sometimes to cause chronic choreoid movements is scarcely relevant, and altogether isolated. The difference between the two conditions is great, and it is possible that a disorder of the action of the cortical cells may be secondary to disease of the thalamus in the one case, and may also occur as a primary change in the other. Whether their affection is or is not the primary one, we can hardly resist the conclusion that the cortical motor cells are deranged in function; and both the frequent mental change, and the pathological alterations that cannot be referred directly to the blood, suggest that there is an extensive affection of the cortex in many cases of acute character. It may well involve the central ganglia, and must necessarily extend to the white substance, the spinal cord, and even the nerves. The functional disturbance can only reach the muscles through the cord and nerves, and a proportionate but secondary derangement of their function must occur.

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* Cases from disease of the crus have been described by Magnan (hæmorrhage), and by Canfield and Putnam (softening).

The alteration in function is evidently peculiar. All functional disorder is attended with nutritional change, and this must occur also in the lower structures that are secondarily affected, whether or not it attains a degree that can be recognised by the microscope. It is indicated in the increased electric irritability of the nerve-fibres of the limbs. But it seems never to reach a destructive degree, or to involve true secondary degeneration. Any destruction of nerve-elements is the result of vascular changes, primary, or secondary to the functional over-action. Moreover this alteration in the nutrition and derangement of function of the nerve-cells tends to pass away when their cause has ceased to act.

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The first important etiological fact is the distinctly predisposing influence of age and sex. Chorea is especially a disease of later childhood; that is, it occurs when the functional development of the nerve-centres has been effected, but their exercise has not yet secured stability of function, and the motor processes are prone to become active independently of deliberate volition. The normal restlessness of childhood manifests the predisposition to chorea. Moreover, the age and sex most liable are also those in which emotion is most active, and exerts the greatest influence on the motor nervous system—in which, indeed, as Sturges has insisted, the physiological expression of emotion is often by movements bearing no distant resemblance to the slighter spasms of chorea. With this predisposition is to be associated the important fact that an emotion has been recognised, by almost all observers, as the one exciting cause that can be traced with frequency. This emotion is alarm, which, throughout the animal kingdom, has a direct effect on the motor centres, essential to the safety of its subject. Were these the only facts of etiology, it would be natural to regard chorea as a purely functional disease, in the strictest sense of the term, a disturbance of the functions of the motor centres depending on their state of functional development, and owning no other cause.

But the association of chorea with acute articular rheumatism is so frequent, and often so close, as to indicate that there is another important element in its pathology. Their close relation is emphasised by the lesion which is common in each, even when they are dissociated—endocarditis, so rare in other maladies, nervous or general. But it is impossible to regard chorea as the result of acute rheumatism, since each disease occurs so frequently without the other, and no close relation has been traced between chorea and the common cause of rheumatism, exposure to cold.

The attempt to explain the relation by assuming that chorea is the result of cerebral embolism is now merely of historical interest. According to this theory, endocardial disease must precede the first symptoms of chorea. But it is only in a very small proportion of the cases that this precedence can be traced. Even in severe and fatal cases, endocarditis has been proved absent after death. In the large number of slight cases in which no sign of endocarditis can be detected we are not justified in assuming its existence. In other cases, again, the signs of endocarditis come on during the course of the chorea. In these two classes, which comprise the majority of the cases of chorea, the embolic theory is quite untenable.*

If the endocarditis is not the cause of chorea it must be either the result of the chorea, or the two must be consequences of a common cause. This conclusion is supported by the fact that, if we are to trust clinical evidence, the endocarditis is often secondary to the chorea in point of time. Pathology at present knows nothing of the direct dependence of such a lesion on an altered state of the nervous system. All known facts regarding the etiology of endocarditis point to its dependence on morbid states of the blood. That excessive muscular action changes the composition of the blood is practically certain, but the endocarditis can scarcely be ascribed to this cause, since it does not occur in other states of muscular over-action; it may be present even in slight cases of chorea, and it may precede the chorea in its development. The hypothesis which seems best to explain the facts is the old theory that the common cause of the endocarditis and the chorea is a blood-state allied to, but not identical with, that which causes acute rheumatism. According to the precise nature of the blood-change, chorea or acute rheumatism or both, with or without endocarditis, may be produced. The facts of these diseases suggest a toxic change of a chemical character, rather than an organised virus. That which causes chorea seems to have no tendency to affect the joints and but little to cause pericarditis, and not to be excited directly by exposure to cold, but it may be accompanied by the condition which causes rheumatism and which presents these features.* Of the mechanism by which the assumed blood-change causes chorea we are still ignorant. The hypothesis suggested by the facts of the disease is that a special influence is exerted on the motor elements of the cortex, deranging their function and producing a disturbance of their nutrition, which, once excited, runs a certain course, varying in severity and duration, but tending to subside. The action of specific blood-states, especially such as are of chemical nature, on certain parts of the nervous system, is well known, and we must remember that, in chorea, the effect is produced on structures that are in a peculiar state of predisposition not only to derangement, but to the particular form of derangement present in this disorder. The disturbance of function seems sometimes to last beyond its cause (since even endocarditis may pass away before the movements), and to leave also a strong disposition to recurrence. The blood-state may act widely on the cortex in some cases, and even on the central ganglia, whose function is still too mysterious to permit us to assign to their derangement a definite part in the pathology of chorea, possibly also on lower motor elements, as in the spinal cord, in less degree. We may note that the excitant of the allied rheumatism, exposure to cold, may act on the peripheral motor structures, and cause multiple neuritis. But the visible changes especially in the vessels, and in prolonged cases in the interstitial tissue, may be in part a consequence of the disturbance of function (see above), since it is certain that they are often absent. They may be also, in part, the effects of the blood-change, which is usually profound in fatal cases,* and to some extent determined in seat by the functional derangement and its secondary effects, while the endocarditis which also usually co-exists apparently gives rise to some of them. If due weight is given to these considerations, there seems little room for the opinion which ascribes the symptoms to these vascular alterations. It requires stronger evidence than is at present forthcoming to prove that conditions that are occasional and variable are causes of the disease, associated, as they always are, with that which seems the probable cause in other cases.

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* That extensive capillary embolism may give rise to movements resembling those of chorea has been proved by Dr. Angel Money ('Med.-Chir. Trans.,' vol. lxviii); but the fact has little bearing on the question of the pathology of human chorea. The facts of disease show that many morbid processes in the brain may give rise to choreoid movements.

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* If the conception of a blood-state allied to, although not the same as, that which causes acute rheumatism seems difficult, we may remember that the varieties in the manifestations of rheumatism must be evidence of a corresponding variety in the constitutional condition that predisposes to it. Of a number of persons who get their feet wet, for instance, one will have a cold in the head, a second acute tonsillitis, a third pericarditis, a fourth acute general arthritis, and a fifth arthritis with endocarditis. The cause is the same in all; the effect must be due to the previous condition of the individual. It does not seem inconceivable that an allied constitutional condition should have an influence on certain nerve-centres, rendering their function and nutrition susceptible of derangement.

We must not, however, regard the blood-change as more than one element in the causation of chorea.† Important facts, already considered, demonstrate not only the influence of functional development as a predisposition, but also that of functional disturbance as an excitant of the disease. The ascertained facts of etiology seem to show how frequent and sometimes how close is the effect of fright in exciting the disease, an agency which has a direct influence on the motor structures ‡ which especially manifest both the predisposition and the developed disease. The violent functional disturbance of this influence seems to involve a shock to their nutrition, which may persist, increase, and run a definite course, most readily in those whose nerve-elements are disposed by neurotic inheritance to instability of nutrition (as well as through developmental instability), or who may possess the deranging influence of the morbid blood-states.

In any theory of the pathology of the disease these three factors must be kept in view, and in any study of the disease the part played by each must be ascertained as far as possible.

The nature of the influence which pregnancy exerts on the occurrence of chorea is still unknown. It is commonly regarded as a reflex influence, analogous to that which causes vomiting; but it is to be observed that the period of development of chorea is usually later, and is much more variable, than that of the morning sickness, and often does not cease immediately on the removal, by parturition, of the source of reflex irritation. We know also very little of the occurrence of chorea from other forms of reflex irritation.

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* Nauwerck ('Beiträge zur path. Anat. u. Phys.,' by Ziegler and Nauwerck, Jena, 1886, p. 407) records an instance of the occasional intense severity of the blood-state and its effects, in a child of seven. Foci of inflammation (mycotic ?) were found in the brain and medulla, and there were also pericarditis, endocarditis, and pneumonia.

† When the causation of a disease is complex, it often happens that, at different periods, attention is too exclusively directed to one of the elements in its production. This seems to be now the case with those who would term chorea an "acute infectious disease."

‡ See above, p. 616; also "Paralysis Agitans."

The influences which determine the precise form that the chorea assumes are also unknown. Maniacal chorea is not met with until the period of puberty, and is very rare in the male sex. It occurs in girls between fourteen and twenty, and not unfrequently during pregnancy,—conditions of age and sex in which emotional disturbance is readily produced.

Thus the chorea of early life is due to a predisposition depending on the state of development and training of the motor centres, varying in intensity according to temperament and age, much increased by the residual effects of a previous attack, and reproduced also as part of the peculiar state of the nervous system during pregnancy. In addition, two other influences may be traced, the rheumatic and the neurotic, each as either a predisposing or exciting influence. They may exist apart or may be combined, the one predisposition with the other excitant, less commonly with both excitants, or both predispositions with one or both exciting causes. In any future investigation it is important that the distinct classes should be separately considered, apart from those that present the combined influences, since facts may be true of one class and not of the other, and be imperceptible when all are taken together.

Diagnosis.—No disease is more easily recognised than chorea in its common form. The peculiar movements at once attract attention; their character is unmistakable; and their recent onset distinguishes the case from the only condition in which similar movements occur—that of infantile disease of the brain. The expression of countenance, with its listless, somewhat vacant aspect, often by itself suggests the nature of the malady. It is only in some special varieties that the affection is less readily recognised. In a slight case of unilateral chorea, if the patient grasps simultaneously the observer's hands, the contrast between the uniform muscular contraction on the one side, and the unsteadiness of the grasp with the affected hand, is readily perceived. In the rare cases in which the legs suffer more than the arms, the interference with standing and walking may resemble that of paraplegia, but the spasmodic movements are always distinct enough to show the nature of the case. A greater difficulty is sometimes presented by the cases of "paralytic chorea," in which there is very little spontaneous spasm, and the loss of power alone attracts the attention of the friends, and sometimes also of the medical attendant. The distinction rests on the facts that the loss of power is confined to one arm, and does not involve the face or leg, and that it comes on gradually (in the course of a few weeks) in later childhood. Close observation reveals slight occasional choreic movements in the weak arm, and often elsewhere, or distinct inco-ordination, especially when the hand is held over the head—an action that often reveals the existence of choreic movements otherwise unperceived. In some cases sustained muscular action is impossible; the grasping fingers, for instance, relax involuntarily, or desired relaxation is for a moment delayed. Once aware of the possible nature of the case, the diagnosis is not difficult. As a rule, when a child between seven and twelve years of age is said to have gradually lost the use of one arm, the disease is chorea.*

Maniacal chorea, when the muscular spasm is slight, may be mistaken for simple mania. There is, however, less continuous garrulity than in simple mania; usually there is some characteristic spasm, and the patients are younger than most subjects of acute mania. The greatest difficulty arises in the rare cases in which the muscular spasm ceases when the mental disturbance becomes considerable, and there is only the history of the spasm to guide the diagnosis.

The diagnosis of hysterical from ordinary chorea rests on the suddenness and isolation of the muscular contractions, on their frequently rhythmical character, on the age and sex of the patient, and, in many cases, on the origin of the disease by imitation. The sudden shock-like character of the contractions is characteristic also of "electrical chorea," described in a separate section.

PROGNOSIS.—In the vast majority of cases, even of severe chorea, the prognosis is favorable. As long as the disease is free from complications, and the patient obtains a fair amount of sleep, no anxiety need be felt, but it is important that the uncertain duration of the disease should be remembered at its onset, and that the parent of a patient should be prepared for it. The chief source of danger is exhaustion from the violence of the movements, and from the deficiency of sleep. The endocarditis has little influence on the immediate prognosis; the chief risk to life that it entails depends on the embolic process, and this is extremely rare. Intercurrent rheumatism is also commonly of a mild type, and the gravest complications of rheumatism—pericarditis and hyperpyrexia—are very seldom met with in chorea. Preceding heart disease renders the prognosis worse in proportion to the gravity of the cardiac lesion, but is rarely a source of anxiety during the attack of chorea itself. The prognosis is worse in the maniacal than in the ordinary form, and considerably worse in the chorea of pregnancy than in that of youth. It is somewhat better in a recurrence than in a first attack, but to this rule the chorea of pregnancy is an exception.

The more severe the disease, the longer will be its probable duration. A recurrence will probably terminate sooner than a first attack. Etiological conditions of age, sex, cause, preceding rheumatism or heart disease, and present endocarditis, afford no indications of the probable duration of the disease. This conclusion is drawn from a careful analysis of the facts of the cases which have come under my own notice. In hysterical chorea the prognosis as to life is absolutely favorable, but the duration of the disease is often long.

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* This statement may seem too absolute, but it is literally true. Organic disease either weakens the leg as well as the arm, or else it causes convulsions or some other symptom that attracts attention, and is mentioned at the outset.

Of the sequelæ of ordinary chorea, mental and muscular weakness always pass away. It is only when there has been severe mania that there is any risk of the persistence of dementia. Convulsive attacks, if of epileptoid type, may continue to recur, and, as we have seen, may develop into epilepsy.

TREATMENT.—The derangement of motor and psychical functions in chorea, its frequent origin in emotion, and the distinct increase of the symptoms that may often be observed to result from emotional disturbance and from mental and physical exertion,—all teach the lesson, which experience confirms, that the first important element in treatment is to secure the patient from causes of mental and physical fatigue and emotional excitement. Lessons should be discontinued, and even in slight cases the periods of physical rest should be increased, only mental or physical occupation that interests without fatiguing should be permitted, its amount depending on the degree and duration of the symptoms. In severe cases there is only too much spontaneous muscular action, and benefit is derived from absolute rest in bed during the height of the disease. Even in moderate cases, the good effect of a few days' absolute rest is most conspicuous, and is often desirable at the commencement of treatment. In such cases, prolonged rest is seldom followed by a continued improvement, and the patient may be allowed to be up during the greater part of the day, provided the movements are not thereby increased in intensity. If the severity of the disease does not make prolonged rest in bed absolutely necessary, the mental depression produced thereby often more than counteracts any good that is done by the rest. All sources of mental irritation should be avoided; a cheerful room and cheerful companionship are most important.

When the movements are severe, extreme care must be taken to obviate the chance of injury from the contact of the limbs with hard objects. These should be removed out of the range of the limbs, or covered with soft padding. The importance of this measure cannot be over-stated. One cause of death in chorea is the effect of slight mechanical injuries, which, in the cachectic state of the patient, often run an unfavorable course, and may lead to blood-poisoning. The risk that the patient may be thrown out of bed by the spasm must not be forgotten. In severe cases it is well to place the patient's bed on the floor in a corner of the room, and to cover the adjacent walls with cushions or mattresses. If bedsores are threatened, a water-bed should be obtained. The influence of the spasm must be borne in mind in all proceedings that are adopted. The temperature, for ment when a patient has been kept constantly asleep by chloral, but this method of treatment is scarcely commended by a case recorded by Bouchut, in which continuous sleep had to be maintained for a month before the movements ceased. In other cases thus treated it has seemed that the period of sleep was merely interpolated in the disease, the course of this being not otherwise modified. The beneficial results have usually been limited to a single case. Inhalations of chloroform have been employed with advantage to secure rest in severe cases, but they have been practically superseded by chloral, which produces the same effect. Antipyrine seems to have a similar action, distinctly useful during the acute stage in many cases, but not when this is over. Five grains may be given three times a day to a child of seven to ten years. Morphia as a sedative is far inferior to chloral. In smaller, stimulant doses (five minims of the liquor three times a day, as advocated by Radcliffe) it seems to be useful in slight cases, but it should not be given if there is mental excitement which may be distinctly increased. Henbane, hyoscyamin, conium, Indian hemp, Calabar bean, cimicifuga, and other sedatives have been employed, but are of doubtful value. Salicylate of soda is sometimes distinctly useful and sometimes fails entirely, a difference that may be found to depend on the associations of the disease and the stage of the attack. It is most likely to be of service when there is endocarditis and a history of rheumatism, family or personal.

Among the so-called nervine tonics, arsenic has long enjoyed a high reputation, which it certainly to some extent deserves. It is usually given by the mouth, and the dose should be gradually increased to ♏x or even ♏xv of the liquor three times a day. It may be combined with morphia (Steiner). The comparative intolerance of the stomach for arsenic has led to its hypodermic injection (Eulenburg, Hammond). By this method much larger doses can be given without unpleasant symptoms, but the emotional disturbance occasioned to children by the operation is a drawback to the method in a disease in which it is so important to maintain emotional tranquillity. In older patients this drawback is of less moment. Hammond recommends, as an initial dose, from five to thirty minims (!) of Fowler's solution, diluted with an equal quantity of glycerine, and, as the best place for the injection, the loose skin on the front of the forearm. Arsenic may be given by the mouth until intolerance is reached, and then continued by hypodermic injection.

Zinc is another drug that has long been praised in the treatment of chorea; both the oxide and sulphate have been given, the dose being slowly increased up to a scruple. Its influence, however, is seldom clear when allowance is made for the long time necessarily occupied in gradual increase of the dose.

Strychnine is of little value in the early stages, but it is often distinctly useful at a later period, especially when the disease lingers on in slight and stationary degree. Calabar bean and eserine ($\frac{1}{25}$ grain) instance, should never be taken in the mouth. I have known a choreic patient to bite off and swallow the bulb of a thermometer placed in the mouth; fortunately the accident was followed by no more serious consequence than an increase in the chorea from the alarm occasioned; the thermometer bulb was safely passed next day *per rectum.*

It is needless to insist on the importance of an adequate supply of easily digested food and of absolute cleanliness. The latter is of great importance when the patient's mental apathy causes unnoticed evacuations; in such a condition the spasm often lessens, and comparative stillness increases the risk of bedsores from pressure. The bowels should be kept open, but purgation is useless.

In many severe cases of chorea the skin is dry and harsh, and in these, free diaphoresis has often a striking effect in lessening the intensity of the disease. The old method was to give a small dose of antimonial wine, and follow this with a hot-air bath, and I have seen excellent results from this treatment. But it is only when the strength is good that the antimony is admissible; the hot-air bath may always be employed if the state of the skin indicates its use.

The influence of drugs on chorea is a subject on which the most diverse opinions have been expressed. It is possible that the conclusions would vary less if the effects of each therapeutical agent were observed separately in the classes indicated at the end of the section on "Pathology." Agents may be frequently useful in one class that have no effect in the other. Many cases of chorea, admitted at an early stage into a well-arranged hospital, and kept at rest, seem to improve as speedily as the nature of the disease will permit, and it is exceedingly difficult, in such cases, by isolated or collective observation and comparison to obtain satisfactory evidence of the influence of drugs on the disease. But this result is not always obtained even in cases and conditions that are apparently promising, while in those that are seen as hospital out-patients, or in which the disease has lasted for a long time, the tendency to spontaneous cessation is much less marked, and recovery is often exceedingly slow. In such cases the influence of drugs is often so distinct as to justify a doubt whether the cases which have given rise to the first conclusion are altogether free from fallacy, and to suggest the desirability of employing those agents which seem most useful, even when their effect is difficult to trace.

Sedatives and tonics have both been largely employed in the treatment of chorea. Of the former, bromide of potassium is of singularly little value. Chloral hydrate is more useful. In severe cases it is often of great service in producing sleep, but in cases of moderate severity its chief use (in my own experience) is to increase the tranquillity obtained by a period of absolute rest in bed at the onset of the treatment. Five or seven grains may be given every six hours for a week or so. Some physicians have described remarkable improvement when a patient has been kept constantly asleep by chloral, but this method of treatment is scarcely commended by a case recorded by Bouchut, in which continuous sleep had to be maintained for a month before the movements ceased. In other cases thus treated it has seemed that the period of sleep was merely interpolated in the disease, the course of this being not otherwise modified. The beneficial results have usually been limited to a single case. Inhalations of chloroform have been employed with advantage to secure rest in severe cases, but they have been practically superseded by chloral, which produces the same effect. Antipyrine seems to have a similar action, distinctly useful during the acute stage in many cases, but not when this is over. Five grains may be given three times a day to a child of seven to ten years. Morphia as a sedative is far inferior to chloral. In smaller, stimulant doses (five minims of the liquor three times a day, as advocated by Radcliffe) it seems to be useful in slight cases, but it should not be given if there is mental excitement which may be distinctly increased. Henbane, hyoscyamin, conium, Indian hemp, Calabar bean, cimicifuga, and other sedatives have been employed, but are of doubtful value. Salicylate of soda is sometimes distinctly useful and sometimes fails entirely, a difference that may be found to depend on the associations of the disease and the stage of the attack. It is most likely to be of service when there is endocarditis and a history of rheumatism, family or personal.

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Strychnine is of little value in the early stages, but it is often distinctly useful at a later period, especially when the disease lingers on in slight and stationary degree. Calabar bean and eserine ($\frac{1}{25}$ grain) have also been recommended, but not on very convincing grounds. Curara has been employed in obstinate and chronic cases with alleged advantage (Diamond, Wright), but it is doubtful whether the employment of so dangerous an agent is ever justifiable.

Electricity has been employed in various ways, especially the voltaic current to the spine, but it is very doubtful whether the agent has any real influence. The spasms are sometimes lessened by the passage of the current through the limbs, but the effect quickly passes away, and I have never observed evidence of permanent benefit. Any effect from the application of magnets is probably psychical and confined to hysterical cases. Massage is chiefly suited to the chronic stage. Another therapeutic measure which has been recommended, but is of very doubtful value, is the freezing of the skin over the spine by ether spray.

Rhythmical movements and mild gymnastics are of service when movements persist as a residual effect of the acute disturbance, or in very slight cases, in which the neurotic element predominates.

CHRONIC ADULT CHOREA

(Hereditary Chorea; Senile Chorea).

Adults after middle life may suffer from an affection characterised by movements closely resembling those of the chorea of early life. It differs, however, in its course, which is not only chronic, but also, as a rule, persistent and progressive. The spasm, indeed, differs a little from the juvenile form in the far slighter degree with which it interferes with voluntary movement, which often stills it. Two forms may be distinguished. One is hereditary, or at least occurs in families, and usually manifests itself soon after middle life, or at least before senility; in early adult life only in cases so rare as to be strikingly exceptional. The other form is isolated, and occurs in old age, somewhat earlier only when the age of the system anticipates the courses of the years. Thus we have hereditary and senile forms, which, however, differ but little in other respects.

Hereditary Chorea; Huntington's Chorea.—In 1872, Huntington of New York described a form of chronic progressive chorea, occurring in families through many generations, among the inhabitants of Long Island, New York Harbour, a favourite haunt of tetanus. It had been familiar to his father and grandfather, who were both practitioners of medicine, as presenting the uniform features of commencement in middle life, chiefly from thirty to forty, affecting the two sexes equally, being associated with mental failure and a tendency to suicide, and continuing until death. If a generation escaped, the malady did not reappear in the family. From the attention the description received, the form is often called Huntington's chorea, but it would seem to have been long familiar to physicians practising in the adjacent districts of the United States. Dunglison* quotes a letter written to him in 1841 by Dr. Waters, of Franklin, New York, giving a description of the malady almost identical with that of Huntington, and also a statement by Gorman, regarding its prevalence in certain parts of Pennsylvania, possibly due to local intermarriage. Since Huntington's description has directed attention to the malady, many instances have been recorded, especially in Germany,† and a few in this country.‡

The affection has been traced through as many as four generations, and in one instance no less than nineteen members of the family were affected (Remak). Of four families descended from a choreic woman, two were healthy, but in the other two, twelve persons became choreic after thirty.§ The two sexes suffer equally. As a rule, it begins without any exciting causes in the middle period of life, or early part of the second half, between thirty-five and fifty. The movements commence gradually in the muscles of the face and upper limbs, and become more severe as they extend. They closely resemble those of ordinary chorea, but may present minor differences. Constant rotation of the eyes has been observed.‖ Articulation may be impossible under excitement, and swallowing reduced to spasmodic efforts. Even the action of the bladder may be deranged. The movements are often arrested by voluntary motion, and they cease during sleep in most cases. The intensity attained varies, but is usually considerable, and constitutes a grave distress to the sufferer. Mental changes are generally associated, especially mental weakness, and hence many of the cases have been reported from asylums. Rarely the mind has been unaffected. Sensation is normal, almost invariably; a loss to pain has, however, been noted. Reflex action is unchanged. The symptoms usually continue until the end of life, which may be near or distant, but one case is on record, seemingly of this form, in which the movements ceased under the influence of arsenic in large doses.¶ When the malady shortens life, it is rather through the indirect influence of the mental state conjoined with the exhausting effect of the spasm, than in consequence of the latter alone.

The morbid appearances that have been found in the brain, and which can be connected directly with the symptoms, seem chiefly to be the result of the over-action of the nerve-elements of the cortex, and do not indicate the primary process of the disease. Slight meningitis, and the accumulation of cell-elements about the vessels and cells, with some increase of the interstitial tissue, have been the chief changes. Nothing is definitely known of the pathology of the disease and its relation to the hereditary causes. It has been thought to be the result of the development of embryonal interstitial tissue-elements, stationary until late life, and then causing irritation of the nerve-structures;[*] but this is a pure conjecture, intrinsically improbable.

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* Dunglison's 'Pract. of Med.,' 3rd ed., Philad., 1848, ii, 216.

† By Ewald, Peretti, Huber, Zacher, Hoffmann, Eichorst, Remak, and others.

‡ West, at Stoke-on-Trent, 'Brit. Med. Journ.,' 1887, i, 435; Suckling, 'Lancet,' 1889.

§ Peretti, 'Berl. klin. Wochensch.,' 1885.

‖ Cazenave, 'Un. méd.,' 1853.

¶ 'Lancet,' 1890, i, 1435.

The isolated senile form, sometimes, although rarely, met with under fifty-five, differs from that last described, not only in the absence of any family tendency, but in the more frequent freedom of the mind, and in the occasional benign course of the affection, which may pass away after lasting for a few months or a year. Usually, however, it continues until the end of life, which it does not always seem to shorten. This variety is less rare in this country than is the hereditary form,[†] to which, indeed, it bears a close resemblance so far as the special symptoms are concerned. The difference does not depend on the age of the sufferer or on the severity or extent of the symptoms.

The malady may begin at any period of late life, even up to eighty years of age, and affects both sexes, males rather more often than in youth. Other neuroses may often be traced in the families of the sufferers, apart from chorea itself. It may come on without obvious excitation, but often follows depressing anxiety. Sudden alarm has preceded the onset only in middle life. A marked difference from the chorea of youth is the absence of any relation to rheumatism or endocarditis; coincident heart disease is degenerative only.

The spasm presents the same irregular movements as in other forms of chorea, and often the same inco-ordination of voluntary movement. The spasm is often most severe, and may render intended actions almost impossible. The affection of the face and tongue may be so extreme as to greatly derange articulation, and to make it almost impossible to understand what the patient says. The spasm is usually increased by emotion, and a patient, who is thrown into the most violent physical agitation by the visit of a stranger, may be comparatively still when free from excitement; sleep as a rule (but not invariably) brings quiet. The affection of the legs is slighter than that of the arms, but may be sufficient to render standing impossible. There may be some loss of muscular power, but it is seldom considerable, so far as the spasm permits the strength to be ascertained.

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[*] Greppin, 'Neurol. Cent.,' Oct., 1892.

[†] Cases have been described by Russell, 'Med. Times and Gaz.,' 1878; in a woman of 74, ending in recovery, by Ferguson, 'Lancet,' 1885; Macleod, 'Journ. of Ment. Science,' July, 1881; McLearn, 'Lancet,' 1885, No. 8; Saundby, 'Lancet,' Nov. 29, 1884 (with references to other cases recorded by Graves, Sinkler, &c.); Suckling, 'Brit. Med. Journ.,' 1888.

The duration of the disease, in the rare cases that have recovered, has been from a few months to one or two years, but that of the persistent form is often long, since it has so little tendency to shorten life. Charcot mentions two cases at the age of seventy-one, in one of which the disease commenced at fifty-nine, and in the other at sixty. In a case recorded by McLearn the disease commenced at forty, and had already lasted fifteen years when the patient came under observation.

In senile chorea changes have been occasionally found in the brain, but frequently the examination has failed to reveal any alterations that can be regarded as the cause of the disease. Slight degenerative changes, such as were found extensively distributed through the central nervous system by Berkeley, in a case of seven years' duration, are probably in part of secondary origin. In one of the cases recorded by Harbinson there was evidence, throughout the brain, of degeneration of the nerve-cells, together with an increase of the lymphoid elements, which were aggregated around the vessels and nerve-cells; this has been the most frequent morbid appearance, combined in cases of long duration with signs of slight general meningitis. In all the three cases recorded by Macleod (see p. 626), in which, however, there was also motor palsy, disease of the motor region of the cortex was found; in two the convolutions were compressed by cystic thickening of the membranes, in the other there were small tumours. Choreoid movements are occasional effects of chronic lesions at the surface of the brain, such as hæmatoma, and some cases of the kind have been erroneously described as examples of simple senile chorea.

The treatment of the two forms of chronic chorea of the adult presents little difference. Rare cases of apparent cure by arsenic and other remedies suitable to the common form, with relief to the symptoms by bromide, cocain, exalgin, antipyrine, and other sedatives, constitute all the results that have been obtained.

"ELECTRICAL CHOREA."

The term "electrical chorea" has long been applied to a peculiar malady that is met with chiefly in Lombardy and some adjacent parts of Italy. The affection resembles chorea in being manifested by spasmodic movements, but differs in the character of the movements and in the course of the disease, and also in the addition of progressive palsy and muscular wasting. The muscular contractions are sudden and shock-like, and thus resemble those that are caused by momentary electrical stimulation. The course of the disease is progressive, and a large proportion of the cases terminate fatally. It is doubtful whether the disease has any resemblance to chorea in nature, and hence it has been called "Dubini's disease," from the physician who first described it in 1846. It affects both sexes and occurs at all ages. Its causes are unknown, but it has been ascribed to some obscure malarial influence, on account of its occurrence chiefly in certain districts. Even in these, however, it appears to be rare. Cases have been described in which the symptoms commenced after a fright, but it is doubtful whether these are of the same nature. The sudden clonic character of the contractions is not, as we have seen, confined to this disease; it is met with in some cases of ordinary chorea, in the hysterical variety, and in some choreoid attacks in adults and the old.* The term has been misapplied, not only to the general clonic spasm of hysterical chorea, but also to the violent local shock-like muscular contractions sometimes met with in that affection, especially in the muscles of the shoulder. Such contractions occur also in the cases described as "paramyoclonus multiplex." But all these forms differ from the Italian malady in their benign and isolated character, which at present seems the most important diagnostic feature.

The shock-like muscular contractions that constitute the first and most prominent symptom of the disease, usually commence gradually in one arm—often in the upper part of the arm—and spread thence, usually to the leg on the same side before they invade the opposite limbs. After a few months or less, the limbs first affected gradually become feeble, the muscles waste, with loss of faradic irritability, and the palsy spreads until it becomes general. In many cases there are epileptiform convulsions, which may be unilateral. In the more acute cases there may be considerable elevation of temperature throughout the course of the disease.

In fatal cases, no constant morbid changes are found in the central nervous system, and not only the nature of the disease, but also the precise part of the nervous system primarily disturbed, is a matter of conjecture. The most plausible theory assumes that the cerebral cortex is the part first affected (and thus the unilateral commencement and convulsions are accounted for), but that the spinal cord suffers secondarily and causes the changes in muscular nutrition and irritability. It has indeed been regarded as the manifestation of a special form of myelitis (Pignacca and Stefanini) due to some peculiar paludal influence. It is doubtful whether any treatment influences the disease, but, from the rarity of the malady, therapeutical experience can accumulate but slowly.

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* The term "electrical chorea" has been applied by some writers to the cases that do not otherwise differ from ordinary chorea. But since it has long been used as a designation for the Italian malady, this use of the word seems undesirable.

"MYOCLONUS MULTIPLEX."

Clonic spasm is a symptom that occurs in various forms and distribution, and constitutes part of the manifestation of many morbid states of the nervous system; and, moreover, these are linked together by intermediate forms, so as to render it difficult to separate them into definite types. Such spasm characterises one form of hysterical chorea, and also the grave "electrical chorea" just described. Besides these, a few years ago Friedreich[*] described, under the name "paramyoclonus multiplex,"[†] a case of clonic spasm affecting the limbs of an adult, and other cases of more or less similar character have been since described.[‡] Males have suffered more often than females. The symptoms have begun at any time of life between puberty and sixty years of age, spontaneously or after a fright, rheumatism, or malarial fever. The last cause may remind us of the Italian "electrical chorea," but myoclonus has not the grave character of the latter. Heredity has not been distinct; when several cases have occurred in a family they have deviated from the common form.

The contractions are sudden and involve the whole or a considerable part of a muscle, often causing an actual movement. They are usually symmetrical, although not equally strong on the two sides, and quickly pass from one part to another. In frequency they vary from ten to fifty a minute, but usually occur in paroxysms, or rather in series, irregular intervals separating the shocks. In some cases, occasional tonic spasms accompany them, in others fibrillary contractions are also observed. The muscles most affected are those of the upper parts of the limbs (especially the deltoid, triceps, biceps, supinator, quadriceps femoris, hamstring and calf muscles) and the facial muscles (chiefly of the lower part), sometimes those of the neck. The muscles of the hands and feet may contract, but seldom cause movement. The diaphragm may be involved, producing a peculiar hiccough, or an odd respiratory sound, in which the larynx probably takes part. The first contractions have usually been in the muscles of the shoulders and upper arms or face. Voluntary movement lessens the spasm in most cases, and may even arrest it. The contractions are also generally diminished by alcohol and increased by mental excitement. The mind has usually been normal, but occasionally there has been some abnormal state, such as the presence of "fixed ideas."

Myotatic irritability has usually been increased; the attempt to obtain the knee-jerk has sometimes excited the spasm, and that in the extensors of the knee may even be provoked by the attempt to obtain the foot-clonus. In one case the movements continued during sleep. In a few cases the clonic spasm has occurred only in paroxysms, occasionally of extreme violence. Thus, in a case recorded by Allen Starr, the movements of the body occasioned by the violence of the spasmodic contractions, alternately in the muscles of the front and back of the trunk, were so violent that "the head was thrown about and the body was tossed about in the chair," and if the attack came on when the patient was walking he was sometimes thrown down. A similar case of paroxysmal clonic spasm was recorded by Pritchard in 1822 as "Convulsive Tremor;" and several others, differing, however, considerably among themselves, have been described by Hammond under the same generic designation. On the other hand, in some recorded cases, the spasm has been trifling in degree, even insufficient to cause any movement of the parts, and only to be seen when the skin was bared, resembling the quivering "live flesh" to which so many persons are liable. It is thus clear that the cases present such differences that it is doubtful whether any should be placed together that do not present the common features of the sudden shock-like character of the muscular contractions, their bilateral symmetry, and the comparative freedom of the extremities. In most of the cases the symptoms have passed away in time, a very marked contrast to the course of those of "electrical chorea." In most instances no morbid changes have been found to explain the condition.

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[*] Friedreich, 'Virchow's Archiv,' Bd. lxxxvi, p. 421.

[†] The prefix "para" is often omitted.

[‡] Seeligmuller, 'Deut. med. Wochenschr.,' 1886; Remak, 'Arch. f. Psych.,' xv; Marie, 'Prog. méd.,' 1886; Homen, 'Arch. de Neurologie,' 1887; Allen Starr, 'Am. Journal of Nervous and Mental Diseases,' July, 1887, and others.

The affection has been regarded by some as closely allied to facial spasm; by others as related to the form of chorea in which the spasm is clonic, and is associated with hysteroid symptoms or other mental changes (forms termed by the French "Tic convulsif" and "Maladies des tics convulsifs"). The disease is perhaps most nearly allied to senile chorea. We have seen that senile chorea sometimes runs a favorable course, and that, even when severe, it seldom causes death. It is doubtful whether the bilateral symmetry of the affection affords any real ground for placing it far from other diseases that it resembles. One curious case has come under my notice in which paroxysms of clonic spasm, somewhat resembling those of myoclonus multiplex, had an irregular distribution.* The most effective treatment has been by nervine tonics, with bromide as a sedative, and the use of voltaic electricity, which has in some way produced a striking result in many cases. A current as strong as the patient can conveniently bear should be passed from the spine to the affected muscles daily for a quarter of an hour. If this fails and the spasm is severe, the hypodermic injection of morphia may be tried, or solanin given by the mouth.

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* The patient was a healthy-looking man, aged thirty-nine, who presented the following condition. His head was in constant movement, jerked to the right, and constantly rotated, as in torticollis, while coincident spasm involved the right shoulder muscles. Slight tonic contraction of the muscles of the face caused the eyes to be partially closed, the eyebrows raised, and the angles of the mouth to be drawn down, and gave to the face an expression of suffering. The least excitement increased the spasm. If he was spoken to, for instance, the clonic spasm extended to the muscles of the forearm and to those of mastication and of the face, changing the expression of pain to one of anguish. When he himself attempted to speak, the resulting disturbance almost baffles description. The jerkings of the arms became most violent, and the head was rotated from side to side with extreme

SALTATORIC SPASM.

The term "saltatoric spasm" is applied to a rare form of clonic spasm in the legs, which comes on when the patient attempts to stand, and causes springing or jumping movements, whence the name.[*] It was first described by Bamberger in 1859, and only a few cases have been recorded.[†] The affection occurs in both sexes, and seems to be rather more frequent in males than in females. The ages of the sufferers have varied between ten and seventy years. In some there has been a history of previous functional nerve-disturbance, epilepsy, hysteria, &c., and in most the onset has been preceded by influences, physical or other, depressing the nervous system. Immediate exciting causes have not, as a rule, been traceable. In a few there have been premonitory symptoms, slight tremor, stiffness, or a feeling of stiffness, in the legs. In one instance the spasm followed a convulsive seizure. The actual onset has generally been sudden. The symptoms consist of alternating contractions in the flexors and extensors of the legs, following each other with great rapidity, and causing jumping movements of such violence that the feet may leave

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rapidity for a few seconds. A similar paroxysm occurred on rising from the recumbent posture in the presence of a stranger; he would stoop for a moment, bend the head as if in expectancy, and then the violent movement occurred. Over the slighter movements he had some control, so that by a great effort he could open the eyes; the eyeballs were usually directed to the right, but he could slowly turn them in any direction. So violent were the paroxysms on an attempt to speak, that for weeks he never uttered a word. He could, however, write fairly well, with occasional interruptions from the spasm. The man's history showed no cause, immediate or remote, for the affection, which had commenced gradually six months before. Under treatment, rest and hypodermic injections of morphia, he gradually improved, and became able to speak to the other patients in the ward, although it was much longer before he became able to speak to a stranger. Ultimately the spasm became trifling, but it had not quite disappeared when he passed from observation.

[*] It has been also called "static reflex spasm" by Erlenmeyer.

[†] Bamberger, 'Wien. med. Wochenschr.,' 1859; Guttmann, 'Berlin. med. Wochenschr.,' 1867, and 'Arch. für Psych.,' Bd. v, 1876; Frey, ib., Bd. vi, 1875; Gowers, 'Lancet,' ii, 1877; Kollmann, 'Deut. med. Wochenschr.,' 1883, No. 40, and 1884, No. 4; Erlenmeyer, 'Cent. f. Nervenkrank.,' 1887.

the floor at each spasm, and the patient is thrown to the ground unless supported. In some cases the spasm has been slighter, and only the heels have been raised from the floor. The spring is the result of spasm, not only in the calf muscles, but also in those of the hip- and knee-joints, while in many instances the muscles of the back have also contracted and have caused a backward inclination of the trunk. This peculiar spasm occurs only on an attempt to stand; as the patient sits or lies down the muscular contractions cease, often entirely, although sometimes a little clonic spasm or rigidity has persisted for a short time. In some cases, pressure on the feet, as the patient lay, caused slight spasm, similar to that produced by an attempt to stand. In no case did the spasm, excited by standing, spread to the arms, but in some instances other voluntary movements, even in the recumbent posture, caused a little clonic spasm, which involved also the arms. Motor power has been normal in most cases, but in some there was trifling weakness after the spasm had existed for some time. Sensation was unaffected in all, but dull pain in the legs, or tenderness of the spine, was present in some of the patients. In one of Bamberger's cases there were other peculiar symptoms,—palpitation, dyspnœa, inequality of pupil, with spasm on one side of the face. In Kollmann's patient other severe disturbances of the nervous system led to death.

The affection has generally continued for some months; in only two cases was its duration less than one month, while in one it continued till the death of the patient, six years after the onset of the symptoms. The cessation of the spasm has, in most cases, been gradual.

The spasm is evidently reflex in character, and has been regarded by most writers on the subject as proceeding from the spinal cord, and the result of a peculiar exalted irritability of the spinal reflex centres, especially of those that subserve the co-ordinated movement of jumping, &c. Freusberg* has shown that alternating contractions in the flexors and extensors of the legs may be co-ordinated in the lumbar enlargement of animals, and I have found evidence of a similar fact in man: in a case of paraplegia, with absolute motor palsy, and evidence of a transverse lesion in the dorsal region, pressure on the soles set up alternate flexion and extension of the hip-, knee-, and ankle-joints, by which the feet were successively drawn up and pushed down.† Apparently the area of increased irritability varies in different cases, since in a few, apart from standing, a peripheral impression has caused movements that spread widely.

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* 'Pfluger's Archiv,' Bd. ix.

† It is to be noted, however, that Woroschiloff (Ludwig's 'Arbeiten,' 1874, p. 110) obtained evidence of a centre in the upper part of the spinal cord of the rabbit, by which springing movements of the legs were apparently co-ordinated. A discussion of some other experimental facts bearing on the subject will be found in my paper on the disease ('Lancet,' 1877).

The disease in most cases has not appeared to be readily influenced by treatment. The spasm has continued in spite of the administration of sedatives; the most important and effective measure appears to be the improvement of the general health, and of the strength of the nervous system, by tonics. The case of shortest duration is one recorded by myself, in which slight but characteristic symptoms (another case having previously been under my observation) were at once cut short, on the second day of their existence, by a copious diaphoresis in a hot-air bath.

HABIT-SPASM.

Children often, and adults sometimes, present spasmodic movements such as winking, twitching the mouth, jerking the head, movements that have a half-voluntary aspect, but which the individuals are unable to control. The patient is said to have "got a trick" of moving the part. This condition has been termed "habit-chorea" by Weir Mitchell, but the term "chorea" is not strictly applicable to it, and "habit-spasm" is, I think, a better name.

The condition is met with chiefly in childhood, especially in the second half, but it sometimes commences in youth, and even in adult life. In young women it is often associated with symptoms of hysteria, and there may be a difficulty in deciding whether certain spasmodic movements are to be regarded as examples of this affection or of hysterical spasm. When it commences in childhood the affection commonly ceases after a few months or years, but it occasionally goes on to middle life or even longer. Rarely it begins late in life, and is then generally permanent. In early life it occurs especially in nervous and excitable children. The affection is said to be more common in females than in males, but it is very often seen in boys. It usually begins between the ages of six and fourteen; I have, however, known it to commence as early as four. Some impairment of general health often precedes the development of the movements; occasionally they appear to be due to some special influence depressing the nervous system, over-work at school, a fright, or some injury. In one case, for instance, the onset followed a fall into the water. They may succeed true chorea, or some local irritation in the part that is the seat of the movements; conjunctivitis, causing blepharospasm, may set up winking movements. In boys the affection is often due to masturbation, and from this cause very severe cases are sometimes met with.

Frequently there is a history of other neuroses in parents or other relatives. More than one child in the same family may suffer, but they seldom present quite the same movements. I have seen, for instance, a different habit-spasm in twin sisters. It is probable that the affection often arises by imitation. There is not often perhaps direct imitation, but the witnessing such movements is apt to produce a peculiar excitability, which finds expression and relief in movements of a similar nature. In cases in which something like direct inheritance can be traced, it is probable that this influence has been at work. A father, for instance, had such movements in the face all through his life, and two of his children likewise presented them. In many cases, however, no causes can be traced, and the affection seems to be the result of the restlessness of childhood, specialised, as it were, in a particular direction.

The movements usually occur at intervals of a few minutes, but sometimes they are almost continuous. Their character varies much in different cases, and even in the same individual. One kind of action, after lasting for a time, may give place to another, or two or three kinds may alternate at the same period. The most common is blinking of the eyelids, by a sudden contraction of the orbicularis palpebrarum, sometimes accompanied with depression of the eyebrows. Another that is very common is a contraction of the zygomatic muscles, moving the angles of the mouth first to one side and then to the other. A movement of the head is also common, a lateral deviation, a rotation, or a nod. In one case there was a backward movement of the head with simultaneous elevation of the eyebrows, as in looking up. In another case spasm at the back of the neck was succeeded by a contraction in the sterno-mastoid. Movement of the arms is also common, shrugging the shoulders, or a peculiar fidgety action of the hand, or some other movement of the arm. One boy would put both his arms forward and then stoop, and he did this under all circumstances, even in church. A movement of the leg is less common; the patient last mentioned afterwards got into the way of kicking his leg forwards in walking, doing so about every dozen steps. Of the twins mentioned above, in one, in whom the spasms began after a fall at three, there was a slight movement of the leg and a twist of the arm, while in the other a slight movement of the head was accompanied by a peculiar action in walking, a half-turn, as if she were looking for something that she had dropped.

Respiratory spasm is also very common; there may be a sudden inspiration, sometimes accompanied by a laryngeal sound, or there may be a sudden audible sniff, or a kind of sob. These may be combined with some other action. Thus, in a boy of eleven, a quick movement of the mouth alternated with closure of the eyes, or with a sudden inspiration that was attended by a slight nasal snore. A peculiar cough is also common in these cases; often it has a laryngeal character, and it may be extremely troublesome. It is occasionally first excited by some laryngeal catarrh and actual cough. One boy began by making a noise in his throat every few minutes, half cry, half cough, so loud that it could be heard outside the house; he said that it was occasioned by a sense of difficulty in breathing. It continued for two years, and then ceased, but soon afterwards twitching commenced in the left side of the mouth, and gradually extended to the whole of the left half of the body; and this continued, varying in degree, for a year. Occasionally these laryngeal sounds are repeated several times in succession. In a case recorded by Blachez the noise made was a piercing cry, compared to the squeak of the child's toy, but much louder.

These habit-movements, as we have already seen, usually cease after a time, but they sometimes persist, and become a source of great annoyance to a patient who has reached adult life. The inability to control them by an effort of the will becomes very marked in such cases. In one instance a girl, who began to blink the eyes in early childhood, still did so at nineteen. In another case, a clergyman of thirty-seven was greatly annoyed by an involuntary smile, of somewhat meaningless aspect, which would cross his face from time to time, without the slightest corresponding emotion, and even when he was engaged in reading in public the most solemn parts of the Church service. It never troubled him when he was preaching or in conversation, but it often occurred when he was looking at another person, and sometimes gave rise to misconception. It commenced at the age of sixteen, and was at first more than a smile, being actual laughter, but it gradually subsided into its permanent form. In another case a lad of eight began to suffer from twitchings, now in one arm, now in the other, two or three quick movements at a time. Such movements persisted, changing their form, until he came under observation at twenty-four, when he presented frequent movements of the face, elevation of the eyebrows, twitching of eyes, &c., and occasionally a sudden up and down movement of the jaw. He could prevent them "when he thought of it," but when his mind was otherwise occupied the spasm increased. Again, a young man, aged twenty-two, presented frequent winking of the eyes and movement of the mouth which had existed for ten years, and had succeeded a peculiar condition that began at ten years old, in which, besides twitching movements of the hands, he had an irresistible impulse to repeat actions; if he had touched an object he felt obliged to touch it again.

These habit-spasms are generally increased by observation, and it is very important that little notice should be taken of them by the friends of the patient. Sometimes the movements will then cease without further treatment. They are seldom under direct voluntary control, and the endeavour to prevent their occurrence may be futile, especially if the attempt is made under the influence of fear of threatened punishment. But the promise of a reward at the close of each day on which the spasm has not occurred will sometimes gradually cause their disappearance; a strong desire, free from any depressing emotion, effects that which the will cannot directly achieve.

Any obvious defect in the general health must be made good, and change of air is often very beneficial, especially when a change in companionship can be secured at the same time. The deterring influence of strangers is often very marked. Of drugs, arsenic has certainly most influence. Weir Mitchell has known the hypodermic injection of arsenic to succeed when other things failed. Nerve-tonics, especially strychnia, may with advantage be alternated with arsenic. If there is much excitability of brain, or if the spasmodic movements are severe, bromide of potassium may be needed, and it is especially useful for the spasmodic cough. Occasionally a local blister is of service, and gymnastics may be employed when the spasm is in the limbs. The smiling clergyman mentioned above ceased to be troubled after he had, for a few weeks, taken some arsenic and iodide of iron, and a dose of bromide each time he had to conduct the service in church.

PARALYSIS AGITANS.

Paralysis Agitans, or Shaking Palsy, is a disease of the second half of life, characterised by the symptoms indicated by its name, muscular weakness and tremor, and also by muscular rigidity. The symptoms usually commence locally and gradually, but tend to spread and to become general. From the fact that it was first fully described by Parkinson in 1817, it has been called "Parkinson's disease," but the name which he gave to it of "shaking palsy" is as apt and adequate* as the designations of most diseases, and both it and its Latin form are firmly established.

Causes.—The disease is about twice as frequent in men as in women (73 males to 42 females in an unselected series of cases that have come under my notice). It usually commences after 40 years of age. Two fifths begin between 50 and 60, and about one fifth in each of the two decades, 40—50 and 60—70; but, on account of the lessened number of persons living, it is probably twice as frequent in the latter as in the former decade. It occasionally begins between 30 and 40, very rarely under 30.† Over 65 it is rare; my series includes two cases (males) beginning at 73, and one (female) at 74.

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* To the name "paralysis agitans" it has been objected by Charcot that either the weakness or tremor is sometimes slight and occurs late, but this fact does not lessen the general applicability of the name, since in the majority of the cases both symptoms are conspicuous.

† Of my cases, the earliest began at twenty-nine, but commencement has been recorded at twenty-five (Hadden), twenty-one (Buzzard), nineteen (Duchenne), and seventeen (Berger).

It is thus essentially a disease of the earlier degenerative period of life. Little variation exists between the sexes in their relative liability at different ages; I have found the average age at commencement to be, in males, 53 years, in females, 51.

Heredity can seldom be traced, perhaps in not more than 15 per cent. In one case the affection commenced, after anxiety, at 33, and was extreme at 39; the patient's father and uncle had suffered from it in late life. This is the proportion in my cases, and also in a series of cases observed by Berger.* The sister of one patient, and the mother of another, and in two other cases the father, suffered also from paralysis agitans. Non-progressive tremor occasionally exists throughout life in some near relation. In other cases there has been a history of insanity or epilepsy in near relatives. The disease has been thought to be more frequent in the "labouring" classes, but the influence of station in life, and also of occupation, is certainly small.

Exciting causes cannot be traced in more than one third of the cases, and vary much in character. The most frequent are emotion, physical injury, and acute disease. Prolonged anxiety and severe emotional shock are the most common antecedents. Sudden alarm may cause general tremor, so that the verb "to tremble" is in process of conversion into a synonym with "to fear." Usually the tremor subsides when the alarm is over, but it has been known to persist and develop into this disease, or the tremor may commence a day or two after the shock. A man was waked by a bell on account of a fire; for a year and a half the same bell always caused transient tremor, which then became permanent, and passed into the typical form of paralysis agitans. It is noteworthy, moreover, that the direction of alarm may localise the commencement of the affection. A remarkable example of this was presented by a woman who, at thirty-seven years of age, was sitting quietly at work, when a stream of water suddenly flowed from a tap on to her left wrist. She was much startled; the left arm immediately began to shake, and the tremor persisted, passing to the leg and afterwards to the limbs on the opposite side. When I saw her, a year later, she presented all the characters of the disease in its typical form. Physical injury is occasionally a distinct excitant, but it must be remembered that this usually involves also emotional shock. That the physical injury is not merely coincident is shown by the fact that the tremor usually commences in the part injured. Thus in two of my cases the exciting cause was a fall on the shoulder, and in each the tremor commenced in the arm injured.* A contusion of the thigh has been followed by tremor in the limb, ultimately becoming general (Charcot). The disease has also followed an injury to the radial nerve (San Martin). Local diseases causing pain sometimes excite it; it began in the leg, in one patient, immediately after sciatica. In a woman of forty-seven it began in the left hand after left-sided pleurisy which needed tapping. Injury may determine the spread of tremor which has already commenced. Thus in one case, recorded by Charcot, dislocation of the jaw, immediately reduced, was followed by tremor in it, which persisted. Traumatic influences sometimes seem to be effective by the concussion of the nerve-centres. In one of the most severe cases I have seen, which commenced at forty, the only traceable cause was a fall from a horse four months before the onset; there was no injury to limb. I have seen one other similar case. Muscular exertion seems to have far less influence than might be anticipated from the effect of prolonged and unaccustomed effort in causing tremor. It seemed to determine the onset in one case in which emotion probably co-operated; a woman was much shocked at a neighbour being killed in a railway accident; she went to the funeral, carrying a heavy child on the left arm; the arm felt very tired afterwards, and the feeling of fatigue persisted and gradually changed to one of stiffness, which proved to be the local commencement of paralysis agitans. Exposure to cold was the apparent cause in one or two recorded instances. Of acute diseases, I have known dysentery and typhoid fever to precede the onset. Malaria is commonly regarded as one of the causes of the affection, but on evidence which is not conclusive. Paralysis agitans is more frequent in England, where ague is almost unknown, than in most malarious countries. I have seen one case in which the disease followed repeated attacks of remittent fever, but the patient was at the same time (during the American war) exposed to great privation and fatigue. Sexual excess is a doubtful cause. Toxic influences which cause general tremor (alcohol, lead, brass-working, &c.) seem to have little, if any, influence in producing true paralysis agitans.

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* In senile maladies the facts that can be ascertained regarding heredity probably fall short of the truth to a greater extent than in the maladies of earlier life, because, as life goes on, the death of older relations lessens the opportunities of ascertaining the facts. It is often astonishing how much disease inquiry sometimes reveals in the families of those who imagine, before the inquiry is made, that they are absolutely free from all morbid heredity, so carefully have unpleasant facts been concealed by those to whom they are known.

SYMPTOMS.—In a well-marked case of this disease, such as is shown in Fig. 148, the aspect of the patient is very characteristic. The head is bent forward, and the expression of the face is anxious and fixed, unchanged by any play of emotion. The arms are slightly flexed at all joints from muscular rigidity, and (the hands especially) are in constant rhythmical movement, which continues when the limbs are at rest so far as the will is concerned. The tremor is usually more marked on one side than on the other. Voluntary movements are performed slowly and with little power. The patient often walks with short quick steps, leaning forward as if about to run.

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* In one curious case the arm in which the symptoms commenced had been the seat of slight tremor since a burn of the palm in youth.

A prodromal stage characterised by rheumatoid and neuralgic pains has preceded the onset in rare cases. Usually the initial symptoms come on very gradually, as tremor, stiffness, or weakness in one hand. The tremor may at first occur only on emotion or fatigue, sometimes only on movement; but it is afterwards constant, and slowly spreads from the part in which it was first noticed.

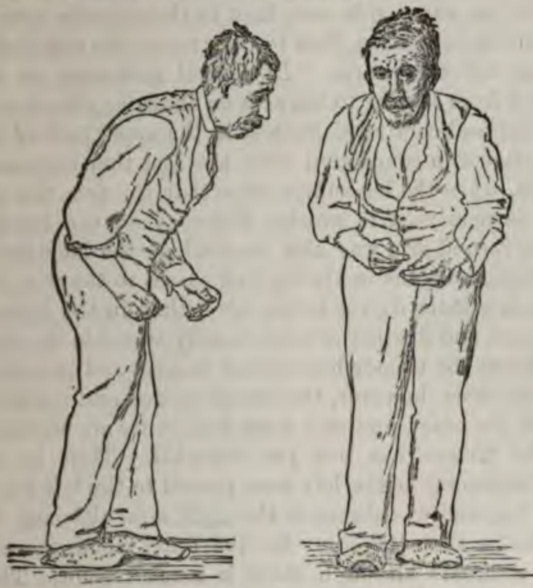

FIG. 148.—Paralysis agitans. (After St. Leger.)

In the majority of cases (two thirds) the tremor is the earliest symptom. Of ninety-two cases in which the mode of onset was noted, it was by tremor alone in sixty-six. Weakness, with or without a sense of "stiffness," precedes tremor in about a fifth of the cases (sixteen cases), and occasionally is conspicuous with tremor from the first (six cases). The commencement is six times as frequently in the arm as in the leg, very seldom in both (arm eighty-six, leg fourteen, both five cases), and more frequently in the left arm than in the right (left fifty, right thirty-six cases).*

In the arm the tremor usually commences in the hand, sometimes in the forefinger and thumb, but I have met with five otherwise typical cases in which it began in the shoulder. In two of these the cause was a fall on the shoulder. From the part first affected the tremor slowly spreads, and the usual mode of extension is from the arm to the leg on the same side, next to the opposite arm, and lastly to the opposite leg. The disease is thus hemiplegic in its progress. Much less commonly the opposite arm is affected before the leg on the same side.* I have known the symptoms to extend to the leg on the same side and thence to the opposite leg, and it has been known to pass from the arm on one side to the leg on the other. The extension from arm to leg on each side is by far the most common course.

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* In my cases the affection began in the two legs with nearly equal frequency, but the numbers are too small to justify the inference that this is the rule.

When the disease begins in the leg, the march of the tremor is less uniform. Usually (as in seven cases) the extension is from the leg to the arm on the same side and then to the opposite arm. In two cases it was to the other leg, then to the arm on the side first affected, and lastly to the other arm. In several instances, in which the tremor passed from the leg to the arm on the same side, it commenced at the shoulder and continued greatest in the upper part of the arm—a point of interest in connection with the fact that not uncommonly in hemiplegia, when the leg suffers more than the arm, the upper part of the arm is paralysed in greater degree than the hand. In one such case the second arm was also invaded at the shoulder. Often, when the affection begins in the leg and passes to the arm, it speedily attains a much greater degree in the latter than in the former.

The weakness and fixation of limb usually succeeds the tremor, and is greatest where the tremor has existed longest and is most marked. In exceptional cases, however, the loss of power and rigidity precede the tremor at the onset, and still more frequently are marked in parts to which the tremor has not yet extended. Thus in one case, the tremor, beginning in the left arm, passed to the left leg and then to the right leg, and was absent in the right arm, although the latter was much weaker than the leg. On the other hand, there is sometimes little weakness although there is much tremor. The several symptoms may now be considered in greater detail.

The *tremor* is an alternating contraction in opposing muscles, causing a rhythmical movement of the parts to which they are attached. It is usually greatest in the hands and fingers, partly from the contraction of the forearm muscles, partly from that in the thenar muscles and interossei; the latter cause a movement of the fingers at the metacarpo-phalangeal joints similar to that by which Orientals beat their small drums. This movement may be chiefly in the thumb and forefinger, as in the act of rolling a small object between their tips. Not rarely the movement is chiefly at the wrist, lateral or antero-posterior, or there is distinct pronation and supination; sometimes the two alternate, or flexor-extensor movements may have a slight lateral direction. The muscles of the upper arm are usually less affected, and those of the shoulder still less. In exceptional cases, as already mentioned, the upper part of the arm is most affected, and the tremor is less in the forearm and still less in the hand. In one such case the chief contractions were in the pectoralis, deltoid, and teres major, slighter in the triceps and biceps, and very slight in the forearm muscles.* In the legs the tremor is usually greatest in the muscles moving the ankle-joint, especially in the calf muscles, and the heel may beat the floor as the patient is sitting; it may closely resemble the contractions of the foot-clonus, though no clonus is obtainable. It is slight in the toes, but may be distinct in the thigh; sometimes in the adductors it is very considerable, rarely in the flexors of the knee. The trunk muscles, especially those of the back, are occasionally involved. I have never met with tremor in the muscles of the abdomen, even when the lumbar muscles were distinctly affected. Usually the head is free from tremor except such as may be communicated to it from the distant oscillation. It does not, however, always escape, as some have asserted. In perfectly characteristic cases of paralysis agitans the head may present conspicuous tremor in consequence of contractions in the trapezius, splenius, and even in the sterno-mastoids,—contractions which may be distinctly felt, and are sometimes considerable, as in a patient with shaking of the arms, stiffness of the legs, and typical aspect; all the neck muscles were involved, but those at the back of the neck in greater degree. I have once met with a slight rotatory tremor of the head due to the deeper muscles.† The masseters are occasionally affected, causing a movement of the jaw, which may amount to one sixth of an inch; the contractions may be greater on the side on which the limbs are most involved. The jaw is usually affected late—after the limbs, but in one case the affection of the jaw came on after the limbs on one side were involved and before the affection spread to those on the other side. Occasionally the tongue is affected; very rarely the muscles of the face. I have once seen distinct persistent tremor in the orbicularis palpebrarum; tremor in the lower facial muscles has been noted by Westphal, and existed in the lower lip and chin muscles (as well as in those of the jaw) in a case in which only one arm was involved. When there has been tremor of the tongue, I have never detected any movement in the soft palate.

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* One such case deviated from the ordinary type in that the upper arm muscles were more affected than those of the forearm, and the intrinsic muscles of the hands were free from spasm.

The movement varies much in its range. It may be so slight as to need close observation to detect it, or may amount to two inches at the extremity of the fingers. It is always slight at the commencement and increases with the progress of the disease.

The time of the movement (according to many tracings that I have taken) varies from about 4·8 to 7 complete oscillations per second (see Fig. 149). It lessens in frequency as it increases in range; the fine tremor of the early stage is often distinctly quicker than the coarser tremor of the later period. Thus in an early case, the very fine tremor varied from 6·4 to 7 oscillations per second; whereas in a later case, with a range of movement of the hand from one to two inches, the frequency was from 5 to 5·4 per second. The tremor in the leg has nearly the same rate as that in the arm in the same case. In one patient, the frequency in the arm was 6 per second, and that in the leg was 6·8. The degree of movement is sometimes very uniform, in others it is somewhat irregular, but it never approaches the irregularity of some other forms of tremor. I have only once observed a tendency to a rhythmical variation.

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* I may remark that in all the exceptional cases mentioned in the text, the diagnosis of the disease was certain; all doubtful cases have been excluded.

† Slight but distinct tremor can, indeed, be felt in the neck muscles not at all rarely. Since I have carefully searched for it, I have found it in no less than eight out of thirty-seven cases.

The great characteristic of the tremor of paralysis agitans is, as Parkinson pointed out, that it continues during rest. The hands go on moving when they are resting on the patient's knee, and the legs when he is sitting. A voluntary movement may stop the tremor for a few seconds, sometimes for many, but it recommences and accompanies the movement. Hence the patient's handwriting reveals his disease; the letters may be fairly formed, but every line is a zigzag. In slight cases the irregularity may be so fine as to need a magnifying glass to recognise it. By an effort the patient can often stop the shaking for a moment, but it then recurs with augmented violence. Although it is the rule for the tremor to continue during rest, the rule is not invariable: exceptions are occasionally met with. In the early stage of the disease, prolonged rest frequently lessens the tremor considerably, and this in cases in which, at a later stage, such rest is without influence. Moreover, in a very early stage of the disease, the tremor may be distinct and even considerable on voluntary movement, and may almost or quite cease as soon as the limbs are at rest.

Lastly, rare cases are met with in which the disease has evidently existed for some time, and yet tremor occurs on movement only. This is especially the case when the fixation of limb preponderates over the tremor. For instance, a woman, aged sixty, had gradually, during four years, passed into a condition in which the features had become expressionless; there were the characteristic posture and slowness of movement to be presently described, considerable weakness of limb, and great sense of heat. During rest there was no conspicuous shaking, and only the finest possible tremor could be discerned in the right, the weaker, hand. But every voluntary movement was attended by regular coarse typical tremor, and this not only in the limbs, but in the masseters, face, and tongue. In another case, with constant tremor in the fingers, tremor at the elbow-joint occurred only on movement. Occasionally the tremor in one hand is constant, and in the other, last affected, occurs only on movement. Fine tremor during rest sometimes becomes coarser during movement. It must, therefore, be remembered that the persistence of the tremor during rest, although usual, is not universal, and its limitation to, or increase by, voluntary movement, does not disprove the indication of other symptoms that the case is one of paralysis agitans.

If the tremor is unilateral, and the shaking of the hand is prevented y force, the other hand, previously free, usually begins to shake. 'he same result may follow the arrest of the movement by an effort f the will, if this can be effected. Sleep usually brings stillness to

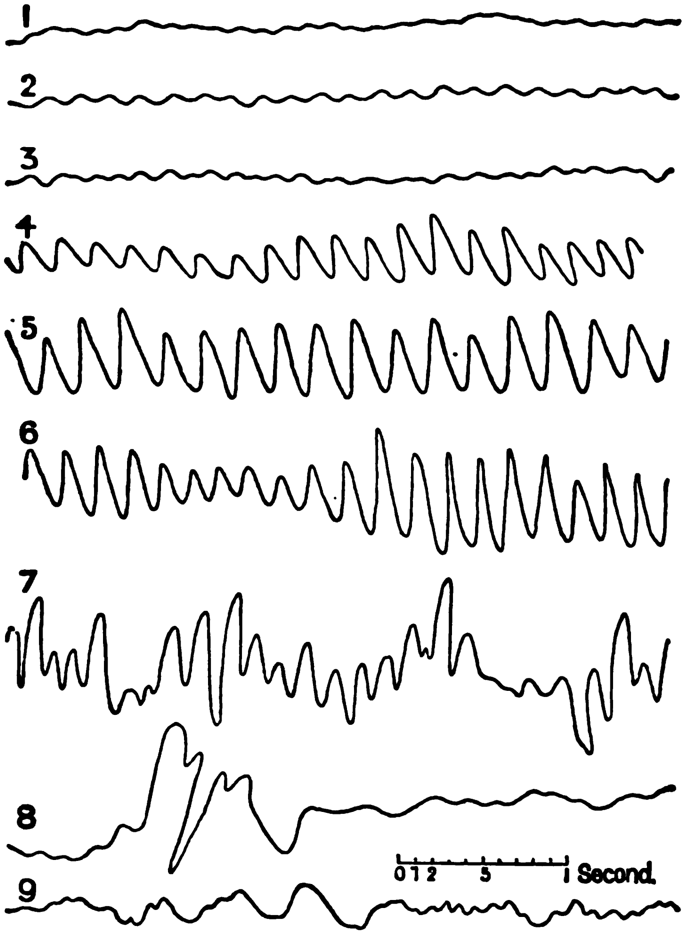

Fig. 149.—Myographic tracings of various forms of tremor taken directly from the moving parts. Reduced to one half.*

1. Tremor after hemiplegia; very fine, rather irregular.
2—6. Paralysis agitans; 2 and 3, very fine tremor; 4, 5, 6, coarse tremor.
7. Insular sclerosis; very irregular in range, although regular in time.
8. General paralysis of the insane.
9. Hysterical tremor.

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* These tracings are fairly typical of a large number I have taken of various rms of tremor.

the shaking limbs, and this although the tremor is considerable; it returns on waking, usually slowly, but on sudden waking from a distressing dream there may be at once severe tremor. Rarely some tremor persists during sleep. I have known the tremor to persist, during sleep, in the arm, and to cease in the leg. In rare cases there are also paroxysmal exacerbations of the tremor in the waking state, excited especially by emotion. The legs in one instance, always stiff, were only at times the seat of tremor.

Muscular weakness and rigidity usually come on together, and are as characteristic of the disease as is the tremor. The loss of power varies much in degree. At first slight, it gradually increases, and is usually greatest in the part in which the tremor developed first and most. The patient may ultimately be unable even to move the index of the dynamometer, or to rise from his seat. But the paralysis is never absolute,—some power always persists. Voluntary movement is not only feeble; it is also slow. It may be slow in execution, or there may be a delay in the commencement of movements that are performed with fair rapidity. This seems to be, in part at least, the result of muscular rigidity, which causes a resistance to passive movement. Another effect of the rigidity is to impress certain characteristic postures on the limbs. These are determined by the fact that the rigidity preponderates in certain muscles, chiefly in the flexors. The arms are flexed at the elbow-joints, sometimes slightly, sometimes almost at a right angle. The wrists are usually slightly extended. The position of the fingers varies; in many cases they are slightly flexed at all joints, in the position that they naturally assume during rest; often they are flexed at the metacarpo-phalangeal joints and extended at the others, from preponderant contraction in the interossei.* There may even be over-extension of the last phalanx, most marked in the thumb, perhaps because the tip of the thumb is pressed against the first finger. The rest-posture is especially frequent when rigidity preponderates over tremor. When the interossei contract in the tremor, the "interosseal posture" is generally very marked. Usually the rigidity can be readily overcome, but in extreme cases (as shown in Figs. 150 and 151) the contracture of the interossei may go on to the degree of permanent shortening, so that the metacarpo-phalangeal joints cannot be passively extended beyond a right angle, just as in contraction of the palmar fascia.† Occasionally only one finger (as the index) is thus affected. In the legs the rigidity involves chiefly the hip- and knee-joints, causing slight flexion of each, and adduction of the thighs. It may extend to the feet, and even cause talipes equino-varus and distortion of the toes—extension of the first and flexion of the other phalanges, so as to cause a claw-like deformity. Permanent contraction of these muscles is very rare, but I have known each foot to be fixed in inversion.

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* In rare cases the digits deviate towards the ulnar side, as in chronic rheumatoid arthritis.

† The contraction in these cases is not of the palmar fascia. Mr. W. Adams was good enough carefully to examine one case for me, and fully confirmed this statement.

The head is usually carried forwards, and the upper part of the spine is bent in the same direction (Fig. 148). In very rare cases there is extension of the spine and neck, and this has even been known to replace the flexion. The facial muscles seem to be unable to respond to the varying changes of emotion; the expression of face is a fixed anxious look, unvaried by smile or frown. In very rare cases, which must be regarded as paralysis agitans, the head is bent strongly backwards. In one such case the affection commenced in the head, but I have seen a similar case in which the arms first suffered. There is sometimes a difficulty in protruding the tongue.

In consequence of the muscular rigidity, certain complex movements are performed in a peculiar and characteristic manner. One of these is walking. The patient rises up slowly from the chair with head and shoulders bent forwards. His steps are short, and the first may be taken slowly and with difficulty, but they become quicker and quicker, until the patient seems to be about to run ("festination"), and often, from the inclination of the body, to be on the point of falling forwards ("propulsion"). It has been thought that this tendency to run is due simply to the forward inclination of the body; the patient, as Trousseau expressed it, has to run after his centre of

FIG. 150.

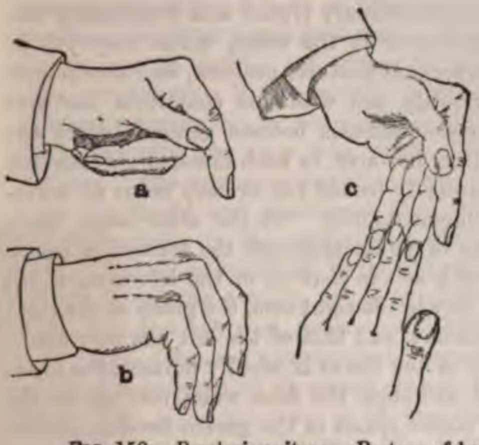

FIG. 151.

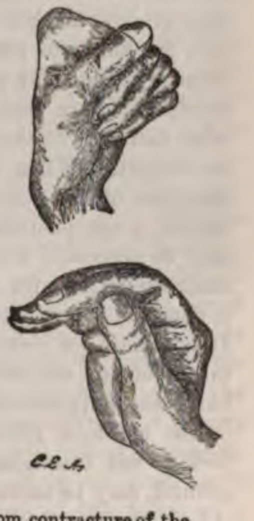

FIG. 150.—Paralysis agitans. Posture of hands from contracture of the interossei: *a*, left hand; *b*, right hand. In the left hand the contracture is greater than in the other, and has led to some permanent shortening of the interossei, so that the fingers cannot be extended even passively. The maximum passive extension is shown at *c*.

FIG. 151.—Contraction of fingers in paralysis agitans. Maximum voluntary extension.

gravity; but that this is not the whole explanation is shown by the fact that some patients, suddenly jerked backwards, may exhibit a tendency to walk backwards which they are unable to avoid ("retropulsion"), and this although they stoop forwards. It is, indeed, not uncommon for patients to be able to walk backwards better than forwards. In one case, an irresistible tendency to run backwards existed only on first rising in the morning. Usually the patient's equilibrium is easily disturbed, and even when they walk fairly well they have considerable difficulty in turning suddenly. One patient, in turning, often took a few unintended steps to one side.

The slowness of muscular action is usually conspicuous in all movements, but it rarely affects the muscles of the eyes. If the patient is to look in a given direction, the eyes are instantly turned, while the head slowly follows them.* Speech has usually well-marked characteristics: the voice is monotonous, being no more varied by emotional expression than is the face. There is a delay in commencing a sentence, but, once commenced, the words are uttered rapidly, often with some confluence of syllables; there is "festination" in speech analogous to that in walking—a condition opposite to the separation of syllables met with in disseminated sclerosis. It is as if the patient tried to speak with a minimum of exertion, and to get his utterance over as soon as possible.

As already stated, although the weakness and rigidity are usually secondary to the tremor, they may come first. We have seen that they precede marked tremor in one sixth of the cases. It is not at all rare for them to precede the tremor in parts secondarily affected. Thus a patient may present the ordinary tremor and weakness in one hand, and there may be no tremor in the other, which may yet be weak and present the characteristic posture, position, and slowness of movement. When the rigidity and weakness constitute the first symptom of the disease, they sometimes become general before the tremor is conspicuous. Often, however, in such cases, slight shaking may be perceived if it is carefully looked for, or may occur on movement, sometimes for a few moments only. On the other hand, there is sometimes very little loss of power although the tremor is grea
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it is in the foot-clonus, no clonus can be obtained by passive flexio
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in these contractions, generally limited to an increase o

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* Debove, however, has noted a retardation in the movement of
if the patient was reading, occasioned a characteristic pause at the
but this is extremely rare.

deformity. Permanent contraction of these muscles is very rare, but I have known each foot to be fixed in inversion.

The head is usually carried forwards, and the upper part of the spine is bent in the same direction (Fig. 148). In very rare cases there is extension of the spine and neck, and this has even been known to replace the flexion. The facial muscles seem to be unable to respond to the varying changes of emotion; the expression of face is a fixed anxious look, unvaried by smile or frown. In very rare cases, which must be regarded as paralysis agitans, the head is bent strongly backwards. In one such case the affection commenced in the head, but I have seen a similar case in which the arms first suffered. There is sometimes a difficulty in protruding the tongue.

In consequence of the muscular rigidity, certain complex movements are performed in a peculiar and characteristic manner. One of these is walking. The patient rises up slowly from the chair with head and shoulders bent forwards. His steps are short, and the first may be taken slowly and with difficulty, but they become quicker and quicker, until the patient seems to be about to run ("festination"), and often, from the inclination of the body, to be on the point of falling forwards ("propulsion"). It has been thought that this tendency to run is due simply to the forward inclination of the body; the patient, as Trousseau expressed it, has to run after his centre of

FIG. 150.

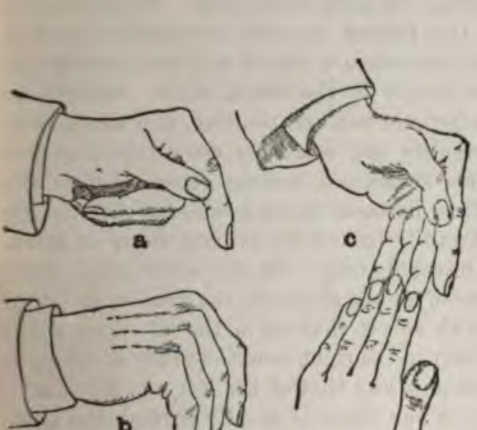
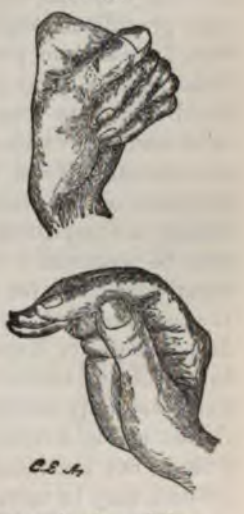


FIG. 151.



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but this is extremely rare.

jerk; in very rare instances a typical foot-clonus can be obtained. The superficial reflex action is also, as a rule, normal. The sphincters are rarely affected; occasionally, in the advanced stage, the power of retaining the urine is weakened, but I have never met with actual incontinence.

The nutrition of the muscles does not suffer until late in the disease. The continuous activity might be expected to lead to hypertrophy, and it has been said to occur, but it is doubtful whether there is ever a real increase in bulk. In most cases, as the disease goes on, and rigidity sets in, the muscles lessen in size, and there may even be conspicuous wasting. The electric irritability of muscles and nerves may be unchanged, but a slight increase may often be detected when the unilateral character of the disease permits comparison of the two sides, and, at a late period, there may be a slight diminution in irritability. I have seen instances of both these changes. The alteration is never considerable, and is always the same to each form of electricity.

Sensory and Vaso-motor Symptoms.—Cutaneous sensibility is never affected in paralysis agitans, but subjective sensations are frequent. Aching pains in the limbs, more or less "rheumatic" in character, are occasionally complained of in the early stage, and they may correspond in seat with the commencing tremor. When the movement is considerable, it occasions a great sense of fatigue, which is accompanied, after a time, by extreme restlessness, and every few minutes some slight change of posture is desired. Frequently also (in three quarters of the cases) there is some abnormal sensation of temperature. The most common is a sense of heat, to which Charcot first directed attention. I have found this to be present in half the cases in which the point was investigated. When slight, it may only cause the patient to dislike hot rooms or many bedclothes; but when more considerable it is a source of much discomfort, and only the thinnest covering can be endured at night, even in the depth of winter. Such sufferers are, as a rule, more comfortable in winter than in summer. The sensation may be referred to the interior of the body or to the limbs, and sometimes it is localised in the most affected limb, especially in the extremity. One patient whose right hand alone shook, complained of burning heat in the palm, passing up the radial side of the forearm to the bend of the elbow. Another, with tremor in the left arm, never would allow this arm to be under the bedclothes. Grasset asserts that this local sense of heat is always the accompaniment of an actual elevation of the peripheral temperature, which is higher than in another individual in the same part; but this is not generally true: the temperature of the body, as a rule, is normal, and I have known actual coolness of the extremities
t with a sense of heat in them.*

ation of heat may be absent, when the tremor is violent,

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wever, once found the affected side (in which there was an intense
° F. warmer than the other.

and it may precede the movement. Thus in one of my patients, the sensation preceded any tremor for four years. In a few cases, instead of a feeling of heat, there is an abnormal sensation of cold; the patient always feels chilly, or there is a special sense of coldness in the affected limbs. Such patients like warmth, and may shake more when cold. Sensations of cold and heat may alternate; in one case, as the disease progressed, the occasional sensations of cold gradually yielded to an enduring sense of warmth, but the alternation may persist even until the late stages; at one time the patient feels cold, and at another bursts into perspiration. Very rarely there is a sense of heat in one part and of coldness in another. It is probable that these sensations, and the local elevation of temperature, are due to vaso-motor conditions.* The sense of heat is very frequently accompanied by increased perspiration, which may be general and profuse on the least exertion of mind or body. One patient could not dictate the simplest letter, even in winter, without first having his coat taken off, so intense was the perspiration caused. Sometimes sweating is local, corresponding to the sense of heat. In one patient, with left-sided agitation, the left axilla was always wet with perspiration, while the right was always dry. This phenomenon sometimes corresponds with another indication of disturbed function of the sympathetic, a small pupil. In unilateral paralysis agitans, I have several times observed the pupil on the affected side to be smaller than on the other, and in one of these cases the patient perspired much on the corresponding side of the head. Frequently both pupils are small, but this may be merely the common senile myosis. They always act to light in uncomplicated cases. Œdema of the legs may exist in the later stages of the disease. The urine is sometimes increased in quantity. According to Chéron it usually contains an excess of phosphates.

The intellect may be unaffected throughout, except by the irritability which usually accompanies the physical restlessness, or by mental depression, which is chiefly the natural result of the physical ailment. Pronounced mental symptoms are occasionally present, however, in the later stages of the disease, especially mental weakness and loss of memory, and they may occur early in its course. If tremor is inconspicuous, they add considerably to the misleading aspect of the case. Very rarely they are accompanied by a tendency to delusions, and occasionally they amount to actual dementia.

Varieties.—The aspect of a case of paralysis agitans varies considerably according to the extent and distribution of the symptoms, which led Marshall Hall to distinguish "hemiplegic" and "paraplegic" forms; and to these a "monoplegic" form, in which only one limb is affected, has been added by Berger. These are not, however, varieties, and should not be described as such; they are merely prolonged stages of a disease which tends to become universal. The aspect of the case is also influenced by the kind of tremor, whether fine or coarse. It also depends on the amount of weakness and fixation of limb, and the relation, in point of time, between this and the tremor, since, as already mentioned, the general aspect, rigidity, position of limb, and muscular weakness may be conspicuous before the tremor is perceptible. It is this feature that gives rise to the best marked variety of the disease. A distinction has been made (by Charcot) between the flexor and extensor types, but it is unimportant on account of the rarity of the latter.

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* It is also possible that the sensation may be due, in part at least, to changes in the sensory centres of the brain, and that the vaso-motor disturbance may be an associated effect of these or other central changes.

Complications.—An attack of ordinary hemiplegia may occur during the course of paralysis agitans without evidence of any closer connection between the two diseases than is involved in the patient's age. As already stated, the hemiplegia arrests the tremor, but, if the paralysis is not permanent, the shaking returns with power of movement, and often becomes much greater than before the palsy, as Parkinson long ago pointed out. It is said that sudden transient hemiplegic weakness may occur from time to time without any lesion being found in the brain to explain it (Berger), and I have known occasional sudden attacks of transient general powerlessness to occur. Early mental failure has been already mentioned. I have once met with convulsive attacks resembling ordinary epilepsy, as a complication of paralysis agitans. The patient was a woman, and both symptoms commenced at the same time, at fifty-nine years of age. Buzzard has recorded a case in which there was a semi-cataleptoid condition of the limbs; when raised they remained so for several minutes, and then slowly fell. Cramp in the legs and soles of the feet is very troublesome in some cases.

Course.—The disease is always chronic, and usually progressive, in its course. The varieties which it presents in its mode of extension have been already described. The rate of progress is sometimes extremely slow. It may remain for two or three years limited to the limb first attacked. Usually, if commencing in the arm, the leg on the same side is involved within two years. Sometimes the extension to the leg takes place in two or three months, and occasionally the arm and leg are affected at the same time. The date of extension to the opposite side varies from six months to three or four years after the onset. The shortest time in which I have known all four limbs to be affected, in a case beginning locally, was nine months. Not unfrequently the tremor is limited to one arm for a considerable time and then rapidly spreads. For instance, in one case the left arm was alone affected for two years, then extension to the leg occurred, and, three months later, to the right arm and leg. In other cases, again, the affection may spread, in the course of a few months, from the arm to the leg on the same side, and two or three years may elapse before the other side suffers. Usually the tremor spreads when it is still moderate in the part first attacked, and there is a gradation of severity in the different limbs, roughly proportioned to the duration of the tremor. Sometimes, however, it becomes severe in the part first attacked before it spreads, or it may be slight in other parts even when general, and intense in the limb in which it began. Very rarely the tremor lessens as the disease advances, and rigidity fixes the limbs. Thus, in one patient, as the second hand became affected, the first became stiff and rigid, and the tremor lessened until it was much less than in the second arm The variations in extension are thus so great as to make it difficult to foretell the course of a commencing case.

Duration and Cause of Death.—Paralysis agitans does not directly cause death, and the advanced age of most of its subjects renders its duration, and its influence in shortening life, difficult to determine. It usually lasts several years. The longest case which has come under my own observation has existed for ten years, but the disease has been known to last for twenty and even for thirty years. Death sometimes occurs from exhaustion, bedsores, &c., in the later stage; more frequently from intercurrent affections, especially of the respiratory organs, facilitated by the progressive muscular weakness, which involves the thoracic muscles as well as others. The tremor has been observed to cease before death.

PATHOLOGICAL ANATOMY.—In most of the cases of paralysis agitans that have been examined by modern methods and by competent observers (Charcot, Westphal, Berger, &c.), no changes have been discovered in the central nervous system or in the sympathetic ganglia; and this although some of the cases were well marked and unilateral, so that the two halves of the nerve-centres could be compared. This fact renders it doubtful whether the changes described by some—for the most part older—observers, had any relation to the disease. An hypertrophy of the nerve-cells of the pons Varolii, which was described by Luys, has not been found by others, and may be dismissed from consideration. An induration of the pons, medulla, and cord was noted by Parkinson, and has since been reported by several observers, but it probably belongs to the changes which alone are found with comparative frequency, those due either to senility or to the indirect consequences of the long disturbance of function, which are without significance as to the seat of its source or the nature of its cause. Among the senile changes is the common wasting of various parts of the cerebral nervous system, cortex, and spinal cord, increase of the interstitial tissue, and degeneration of the arteries. These are all, however, frequently absent. Especial weight has been attached to interstitial sclerosis of the peripheral nerves and traces of chronic changes in the muscles, supposed to suggest inflammation in them; but these may reasonably be regarded as among the second class of changes, those that result from the long-continued functional disturbance.*

* For some recent observations on the pathological histology of the disease see Koller, 'Virchow's Arch.,' Bd. cxxv, and Ketscher, 'Neur. Cent.,' March, 1893.

PATHOLOGY.—In the absence of any anatomical evidence of the seat and nature of the disease, the pathology of paralysis agitans has been the subject of copious speculation, into much of which it is not profitable to enter at length. The facts of the disease suggest, however, certain conclusions which may be briefly pointed out. We are justified in regarding the affection as one of the central nervous system, since we know nothing of clonic spasm in whole muscles (and emphatically nothing of such contractions alternating in opposing muscles), in consequence of a primary disease of the muscles themselves, or in consequence of idiopathic disease of the nerves. Such clonic contractions must be ascribed to the abnormal action of nerve-cells. Tonic contractures may be the result of primary muscular changes, but they also are more commonly the result of a morbid state of the nerve-centres, and the correspondence in seat of the clonic spasm and contracture of paralysis agitans makes it almost certain that both are due to the same cause; the fact that tonic contracture may precede clonic spasm prevents us from regarding the former as the result of muscular changes induced by the latter. That the morbid action is not primarily in the spinal cord is probable from the facts that, as a rule, even when severe, the tremor ceases during sleep, and that the unilateral commencement and hemiplegic extension are unlike those of spinal cord disease. The regions which have been thought to be the seat of the morbid process, by different authorities, are the pons, the corpora quadrigemina, and the cerebellum. Regarding the two former, the theories are purely speculative. We know at present little of the function of the large amount of grey matter which is interspersed among the fibrous tracts of the pons, or the symptoms of its disease; but the effects of lesions, degenerative or acute, of other parts of the pons lend no support to the hypothesis. Nor have symptoms in any way resembling paralysis agitans been observed in disease of the corpora quadrigemina. The theory that regards the cerebellum as the part affected is due to Dr. Hughlings Jackson, but the only actual fact on which it is based is that in some rare cases of cerebellar disease the interosseal position of the hands has been conspicuous. This has led him to suppose that there is a normal antagonism between the cerebral and cerebellar influence, the former tending to cause flexion of all the phalangeal joints, the latter flexion of the metacarpo-phalangeal and extension of the others by the interossei. But the symptom is extremely rare in cerebellar disease; as a rule, it is absent, and, by the converse process of reasoning, it might with equal cogency be ascribed to unantagonised cerebral influence. The same posture of hand is seen in tetany, in many cases of post-hemiplegic disorder of movement, and, what is less equivocal, it is a common posture of the hand during epileptic convulsions, in which the excess of nerve force is certainly not evolved from the cerebellum. If we turn to another class of facts, the cases in which tremor results from organic disease of the nervous system, we find the disease situated, as a rule, within the cerebral hemispheres, in the optic thalamus, posterior part of the internal capsule, foot of the corona radiata (Nothnagel), parietal lobe of the cortex (Chvostek), and island of Reil (Leyden).

In considering further the indications afforded by the symptoms as to the part of the brain that is the seat of the primary disease, the first obvious considerations are those that are mentioned in the discussion of the pathology of chorea, and are equally applicable to paralysis agitans.

That man, as regards his physical frame, is a member of the animal world is generally admitted, and makes it instructive to trace the indications of the relationship in disease and its causation. Tremor is an effect of fear in animals as in man, and doubtless depends upon a relation between the centres concerned in the emotion and those which produce the motion needed to save from danger, and which underlies the meaning of the original word. It is when the motion cannot take place that the influence on the motor centres deranges their action in the manner that is manifested as spontaneous tremor (see under Exophthalmic Goitre). The profound disturbance may even be associated, in man, with an inhibition of voluntary motion, as when a person is "paralysed by fear," an expression by no means merely symbolical. Such a disturbance must have a prolonged influence on the nutrition of the disturbed structures, and it is, therefore, not surprising that it should lead to disease in those who are predisposed—in the old to paralysis agitans, in the still unstable centres of the young to chorea. Such considerations lead us to regard the motor centres of the cortex as being, in both diseases, the seat of the derangement on which the symptoms depend. If we ascend the motor path from the spinal cord, the first nerve-cells we meet are those of the motor cortex; and if the stimulation causing the tremor comes from the brain, it must proceed immediately from these cortical nerve-cells. That it does so is rendered additionally probable by the fact that, as I have observed, an attack of cerebral hemiplegia at once arrests the spasm on the affected side.

The question then presents itself—Are not these cortical cells the primary seat of the disease? The early symptoms of paralysis agitans are almost exclusively motor, and there is nothing in them that is inconsistent with their origin in this part of the cortex. Their peculiarities must, on any hypothesis, be due less to the seat than to the nature of the morbid process. The only sensory symptoms that are present are secondary in time and inconstant in occurrence; they are, therefore, probably secondary in their origin and distinct in their source. We do not at present know on what they depend, whether the abnormal sense of temperature is due to derangement of the sympathetic, or is the result of a change in the sensory centres in the brain. In the occasional inequality of the pupil, which is smaller on the most affected side, we have evidence of diminished action of the sympathetic; and the local increase in temperature, occasionally observed, may have a similar significance. But we know of no mechanism by which the motor symptoms can arise from a primary derangement of the sympathetic, and it is probable that this is simply secondary. It is certain that the functions of the sympathetic are represented in the cortex, and may be deranged by cortical disease. Lastly, it may be remarked that the tremor of fear just referred to* is most readily conceived as of cortical origin. At the same time it must be remembered that all disturbed function of high motor centres is expressed through lower centres (*e. g.* through the motor nerve-cells of the spinal cord), the functions of which are thus disturbed in a similar though secondary manner, and such secondary disturbance, when long continued, may tend to acquire relative independence.

The slight change in the electric irritability of the peripheral nerves may have the same significance as in chorea, and the remarks on p. 600 are equally applicable to paralysis agitans. The structural changes in the muscles present in rare cases are no doubt merely the result of their long-continued active contraction.

The precise character of the motor disturbance in shaking palsy must depend on the nature of the morbid process. This is apparently one that leads to a treble change. There is the intermitting release of nerve-force, causing the tremor; there is a more continuous and slighter activity of the cells, producing the rigidity; and there is lessened capacity for activity, causing the weakness. It is possible that these are the result of slight differences in the precise character of the morbid change in the same cells. What that change is we have as yet no indication. The persistence of the disturbance of function shows that it must depend on changes in nutrition, but these are apparently far too fine to be revealed by alterations of structure recognisable by the microscope. Their relation to age as well as their character justifies us in regarding them as "degenerative," while their character on the one hand, and the negative results of microscopical research on the other, alike show that the alterations in nutrition must begin in the nerve-elements themselves. Tremor no doubt depends immediately on physiological arrangements in the nervous system, which determine the relative action of antagonistic muscles. We have already considered this question in connection with the pathology of nystagmus. Every contraction of one set of muscles involves a related contraction of their opponents, essential for the steadiness of muscular action and facilitating the alternation of movements; it is easy to conceive that tremor may result from its derangement.

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* In connection with the probable source of the movement, a curious symptom described by one patient may be mentioned. There was tremor in the arm, and rigidity in the leg with a constant feeling that it was going to shake.

DIAGNOSIS.—When its symptoms are well marked, no disease is more easily recognised than paralysis agitans. The patient's aspect, carriage of body and limb, and continuous movement make up an unmistakable picture. A difficulty arises chiefly in cases in which the tremor is absent or quite indistinct, and the evidence of the disease consists only of the loss of power and the fixity of feature and of limb, the slowness of movement, and the forward stoop. A knowledge of the significance of these symptoms, which are as characteristic as is the tremor, will prevent error. It is important that their aspect (as distinct when tremor is present as when it is absent) should be firmly fixed in the student's memory. The greatest difficulty is when weakness and stiffness of the limbs come on without the usual attitude and facial expression, and especially if there is also chronic mental failure. I have known the nature of such a case to be either mistaken or mysterious during many years. The absence of increase in the myotatic irritability constitutes a difference from most other diseases causing similar weakness; but, as a rule, the aspect of the patient and mode of movement are at once distinctive.

In some cases the weakness of the legs is the chief source of inconvenience to the patient, and the case may be thought at first to be one of disease of the spinal cord; but attention to the state of the arms and face will generally show the nature of the case, and a knowledge of the occasional limitation of the symptoms to weakness and rigidity of the legs is sufficient to prevent a mistake.

There are certain other diseases, however, from which there may be a difficulty in distinguishing cases that are not very well marked. Old persons sometimes present fine tremor of limb which bears considerable resemblance to that of commencing paralysis agitans, but does not present the same tendency to increase, or the associated rigidity. This "senile tremor" occurs chiefly in extreme old age, while paralysis agitans usually commences somewhat earlier. In senile tremor the head is especially affected, and often is the part in which the shaking commences, while in paralysis agitans it usually escapes, and its affection is seldom considerable. Senile tremor often commences on both sides, paralysis agitans almost always on one. There is not, however, a sharp line of demarcation between the two affections, and it may be difficult to say in which category some cases should be placed. Moreover, simple tremor sometimes exists throughout life, especially in males, beginning about the time of puberty and persisting with only slight variations in degree. When such a patient reaches the late period of life, he may be thought to be the subject of paralysis agitans, but the other symptoms of this disorder are absent, the tremor is less regular, affects the head in conspicuous degree, and the history of the two affections is different. Nevertheless it is possible that some examples have been published as instances of paralysis agitans beginning in early life.

In "disseminated" or "insular" sclerosis, tremor occurs on movement only, and is usually more jerky and irregular in character than in paralysis agitans. In the cases of shaking palsy in which the tremor ceases during rest, there are the general aspect, carriage, and rigidity which are absent in disseminated sclerosis. Moreover, insular sclerosis usually occurs before, paralysis agitans after, thirty-five. In the former the head is usually conspicuously affected, and there is nystagmus, a symptom unknown in the latter. In sclerosis, articulation is syllabic; in paralysis agitans the words are uttered quickly, and there is a tendency to run them together, rather than to separate the syllables. In the majority of cases these indications will enable the diagnosis to be made without difficulty. In very rare cases (Herterich, Schultze) insular sclerosis is said to have been found after death, although the symptoms of paralysis agitans were present during life, but the precise nature of these cases is not clear; we have seen how variable and even equivocal may be the manifestations of insular sclerosis, and a fine tremor is alone of little diagnostic value. The cases are, moreover, too rare to be of practical diagnostic importance.

The tremor that succeeds hemiplegia is fine, and is limited to the seat of paralysis, of which there is the history to guide the diagnosis. The differences between clonic retrocollic spasm* and paralysis agitans might seem to preclude the possibility of error. But the rare cases of paralysis agitans in which the head is carried backwards may be mistaken for retrocollic spasm, and the finer clonic form of retrocollic spasm may also (as I have known) be mistaken for paralysis agitans. The diagnosis rests on the characteristic affection of the hands in paralysis agitans, which commonly precedes the affection of the head, on the limitation of the spasm to the muscles moving the head in retrocollis, and on the fact that in this disease there are contractions in the frontal muscles, synchronous with those at the back of the neck.

In all cases of paralysis agitans the general posture and carriage of the patient are of great diagnostic importance, since they are seldom absent, are easily recognised, and probably never mislead.

The PROGNOSIS of a typical case of paralysis agitans is very unfavorable, so far as recovery is concerned. The disease seems to consist in degenerative changes, which are usually as incapable of arrest as are the advancing years. A trifling amelioration or retardation of progress is all that can be hoped for. When the symptoms depart from the ordinary type, the prognosis is so far better in that there is a little more chance of doing good, since in a few of these cases remarkable improvement has been obtained by treatment. The danger to life, however, is almost as small as is the chance of recovery. The disease always lasts several years, and its duration is likely to be the longer, the slower its advance. The more the case approaches to senile tremor,—that is, the finer the movement and the slighter the tendency to rigidity, the less is the disease likely to interfere with the patient's comfort.

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* The form of wry-neck in which the muscles at the back of the neck are involved on both sides.

TREATMENT.—A malady that depends on changes in the finer nutrition of the nerve-elements, which have no tendency to proceed to the degree of destructive degeneration, might be expected to be amenable to treatment, were it not for the persistent character of the disturbance of function that is induced, and for the commonly senile nature of the change. The absence of spontaneous variations and of spontaneous subsidence seems to indicate alterations of a character which resists all attempts to induce their subsidence, and this is not surprising if they are, to a large extent, the expression of the influence of age, or of the same state produced before senility by influences that have an equivalent effect. For the most part the object of treatment has therefore to be merely relief or retardation.

All causes of mental strain and of physical exhaustion should, as far as possible, be prevented. Life should be quiet and regular, freed, as far as may be, from care and work. All treatment that may fatigue or exhaust the patient's strength should be avoided. Among internal remedies, all varieties of nerve sedatives have been employed, and each has been praised by some and found to fail by others. Many of them, especially morphia, conium (Berger), hyoscyamin (Charcot and Oulmont), hyoscin, solanin, and Indian hemp, quiet the tremor for a time. Of tonics that have been recommended, carbonate of iron (Parkinson), chloride of barium (Brown-Séquard), and strychnine have seldom been found, on extended trial, to deserve confidence; the last, however, occasionally seems to exert a good influence, and it has been found to lower the motor function of the cortex of the brain when it exalts that of the spinal cells. Arsenic, by the mouth, occasionally seems to do some real good; its subcutaneous use has been recommended by Eulenburg. Curara and bromides are valueless. My own experience is to the effect that arsenic and Indian hemp, the latter sometimes combined with opium, are of most use. I have several times seen a distinct improvement for a considerable time under their use. In one case tremor had commenced in the right arm and leg an hour after a railway accident, and extended, three months later, to the left arm. Two years subsequently there was constant lateral movement at the wrist-joints, but no tremor of the fingers. A great improvement occurred on Indian hemp, and a year later the tremor had almost ceased, being occasional only. It is in cases which, like this one, present some deviation from the common type that benefit is most frequently experienced. Electricity in all forms is useless. Static electricity was suggested long ago by Reynolds, and later by Charcot. I have tried it in several cases very thoroughly, but could not observe improvement from its use. So, too, with voltaic electricity. I can entirely confirm the conclusion of

Berger, who treated twenty cases sedulously in this manner without any of them being in the least improved by it. Nerve-stretching has been employed, but without any enduring result (Westphal). In one case stretching the brachial plexus is said to have lessened the tremor in the leg and removed it in the arm, but it caused paralysis of the limb with extensive wasting of the muscles. Considerable transient relief is given by very gentle superficial hand-rubbing of the part which is the seat of the tremor; this is markedly lessened for the time, and the patients express themselves as much more comfortable for it. Although not permanent, the temporary effect is not to be despised in advanced and severe cases. Charcot has recently suggested the employment of a "shaking seat" as a mode of treatment, on the ground that some patients describe relief after riding in a conveyance which causes vibration, but this is certainly not the usual experience of sufferers from this disease.

OTHER FORMS OF TREMOR.

Senile Tremor.—In extreme old age slight tremor is often observed without the muscular weakness and rigidity that occur in paralysis agitans. At first it is noticed only on voluntary movement, and is generally influenced to a greater extent by movement than is paralysis agitans, ceasing or almost ceasing during rest, and always passing away during sleep. It usually commences in the arm, and often in both arms at the same time, but the head is affected early in the course of the symptoms much more frequently than in shaking palsy, and occasionally the tremor begins in the muscles of the neck. The tremor is always fine, the range of movement being very small. After a time it may occur during rest as well as on movement. It is little influenced by treatment. As already stated, it is doubtful whether this senile tremor is far removed from paralysis agitans, although, as a rule, the other symptoms of shaking palsy remain absent. Some cases are met with of a character intermediate between the two affections, and both may occur in different members of the same family.

Simple Tremor.—Young or middle-aged persons, of both sexes, sometimes become affected with tremor, generally fine but sometimes irregular and unequal in the degree of movement, unaccompanied by weakness or rigidity. It is usually excited by movement and increased by emotion, while it commonly ceases during rest. The will can often control it for a time, and it interferes much less with complex actions than does the tremor of paralysis agitans. The handwriting, for instance, rarely exhibits irregularity. The parts affected are chiefly the hands and head, but the muscles of the face and tongue often present irregular tremor on movement, and the patient's aspect may closely resemble that of one affected with delirium tremens, general paralysis of the insane, or the slighter tremor of chronic alcoholism.

By its resemblance to the last it often gives rise to misconception regarding the habits of the sufferer. The shaking, in rare cases, disappears for a time, spontaneously or as the result of treatment, but it usually persists, getting neither better nor worse, to the end of the patient's life; on the duration of this, and on his health, it seems to exert no influence. Rarely it has commenced in early or middle adult life, and, after lasting for a few years, has ceased.

The causes of this simple tremor are generally obscure. It is sometimes apparently first excited by emotion, more frequently by prolonged anxiety, but still more frequently no direct cause can be traced. Its occurrence is, however, distinctly favoured by an inherited neuropathic tendency, and there may even be direct inheritance of the special form, or several collateral members of a family may suffer from it. An hereditary relation to paralysis agitans is sometimes to be traced, but only in rare instances; a case has been mentioned in the account of that disease in which one member of a family suffered from it, and another from simple tremor. Two remarkable instances of this inheritance have been related by Liegey.* A gentleman suffered throughout his life from tremor, and died at eighty-two; his sister also trembled, and his mother, after a mental shock, presented tremor during the latter part of her life. The other case illustrated the occasional affection of collaterals, since four members of the same family suffered from tremor of hands and lips, interfering with speech. In three it commenced in early adult life, but in one, a female, not until the cessation of the menses. A daughter of the latter suffered from symptoms resembling those of disseminated sclerosis.

Nothing is known of the pathological anatomy of the affection, and it is unlikely that anything will be ascertained. General considerations lead us to refer it to a deranged action of the motor nerve-cells, either in the cortex of the brain or in the lower centres of the medulla oblongata and spinal cord. We cannot refer it to the peripheral nerves, since their proved disease has no such symptom for its consequence. It is possible that it may be due to altered function of the cerebellum or of some of the basal ganglia of the brain, but the facts to justify such an hypothesis have yet to be ascertained.

This simple tremor has no consequences beyond its inconvenience. It does not, however, prevent the subject from pursuing his occupation as efficiently as any other person. In spite of the occasional collateral relation to paralysis agitans, it seems to have little tendency to develop into the latter disease, and certainly is less closely connected with it than is senile tremor. When it has lasted for several years it is seldom influenced by treatment. In early cases, the treatment recommended for paralysis agitans best deserves a trial, but even in them the affection is remarkably intractable.

Hysterical tremor is very common. It may occur in two forms, one fine, the other coarse. The latter often consists in a rhythmical movement of the hands or head, continuing apart from any voluntary effort. The fine tremor occurs especially on movement, and its characteristic is irregularity (see Fig. 149, tracing 9). It varies in degree from time to time, presents a transient, often jerky increase in range, or acceleration in time, which are almost pathognomonic. Its characters and treatment are described more fully in the chapter on hysteria.

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* 'Journal de Méd. de Bruxelles,' 1882.

Asthenic Tremor.—In simple weakness, such as follows an acute disease, muscular tremor is common. It is fine in range, occurs only on movement, and is usually most marked after some slight exertion, as an effect of fatigue. The muscular weakness, the conspicuous general debility, the distinct cause of the asthenia, and the usual absence of the tremor, for a time, after rest, render the nature of the case clear. We do not know whether it depends on imperfect nutrition of the muscles or of the motor nerve-cells; the symptoms can be explained on either hypothesis.

Toxic Tremor.—In mercurial poisoning, tremor is the conspicuous symptom, and also in some cases of lead poisoning and in chronic poisoning by alcohol, opium, and chloral. These forms need not be further considered here.

WRY-NECK: TORTICOLLIS.

The term "torticollis" or "wry-neck" is applied to those conditions in which contraction of the muscles of the neck causes an unnatural position of the head. The contraction may be a permanent shortening of a muscle, or it may be an active spasm in one or several muscles. Besides these two chief varieties, a similar effect may be due to two other causes, which are chiefly important in respect to diagnosis, and may therefore be briefly mentioned. One is simple rheumatism of the muscles of the neck, impeding movement. The "stiff neck" of adults scarcely merits the distinctive name of wry-neck, but in children more pronounced deviation to one side or backwards is occasionally met with. The affection follows exposure to cold, and is marked by considerable tenderness of the cervical muscles, which is clearly the chief cause of the unnatural position. These cases are really rheumatic and not nervous in nature, and have been already alluded to in the account of the diagnosis of meningitis. The second condition is one that may be termed "false torticollis," because the deviation of the head depends, not on the state of the muscles, but on some other cause. The deviation, however, usually puts the sterno-mastoid on the stretch, and may thus give rise to an erroneous impression that the muscle is contracted, and is the cause of the deviation. The most common cause of this form is disease of the cervical vertebræ. The distinction from the true form will be considered in the section on diagnosis.

FIXED WRY-NECK: CONGENITAL TORTICOLLIS.

Fixed wry-neck depends upon shortening of some muscle, almost always the sterno-mastoid, which is often also atrophied, and sometimes firmer than normal. The condition is met with chiefly in children, and is thought to depend, in some cases, upon injury to the muscle from the traction on the neck that occurs during birth (Stromeyer). In other cases it is supposed to be due to developmental shortening of the muscle, the result of the inclined position of the head of the child in the pelvis.* It may not be noticed for some time after birth, in consequence of the shortness of the neck of young children. A similar condition may arise in adult life from an injury to the muscle, leading to secondary inflammation, and cicatricial contraction.

In fixed wry-neck the head deviates a little to one side, in the manner to be presently described as characteristic of active shortening of the sterno-mastoid, and it cannot be turned towards the side on which the muscle is contracted. The cause of the deviation is at once seen when the neck is examined; the sterno-mastoid, on the side towards which the head cannot be turned, is rigid, and stands out conspicuously; there is no active spasm. The affection is one of small importance. It can be readily and permanently cured by division of the tendon of the contracted muscle. When secondary changes have taken place in the articulations, it may be necessary to keep the head in a proper position, for a time, by some support.

SPASMODIC WRY-NECK.

Spasmodic wry-neck is a very different affection from that just described, although the two have been sometimes confused in descriptions of the disorder. The spasmodic form is a malady analogous to the facial spasm that has been described in the section on diseases of the facial nerve.

It is necessary to consider together all forms of active spasm in the muscles of the neck. Neither the character of the spasm, nor its distribution, affords ground for the separation of the varieties that it presents. The spasm may be either tonic or clonic, and both kinds of spasm may be met with in the same case, and even at the same time. The most important difference in the distribution of the spasm is, that while in most cases the spasm is either confined to one side, or is so much greater on one side as to cause a lateral deviation of the head, in a few cases the spasm is equal on the two sides, and is either confined to, or greater in, the muscles at the back of the neck, so as to produce a backward movement of the head. The second variety may conveniently be distinguished as "retrocollic spasm."

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* It is much more frequent on the right side, which gives support to the theory, as Busch points out, since in 70 cases out of 100 the head is in the "first position," which entails an inclination such as would permit the right sterno-mastoid to shorten.

Etiology.—Spasmodic torticollis is more common in women than in men. Of thirty-two cases, of which I have notes (all those of hysterical nature being excluded), ten were in males and twenty-two in females. It commonly begins in the middle period of adult life, between thirty and fifty years of age. Two thirds of the cases commence during this period; a few commence between twenty-five and thirty.* It occasionally begins after fifty; the latest case that has come under my notice began at sixty-six. Both sexes seem to suffer in about the same proportion at the different ages. Cases in females under thirty are often of hysterical nature, and this is also probably true of the rare cases in which similar spasm is met with in boys.† In rare cases, torticollis, of peculiar and irregular forms, occurs in infants, chiefly towards the end of the first year of life. It usually ceases after a few months' duration. Still more rarely it begins in childhood and persists, as in the case of a boy in whom it commenced at the age of four and was still severe at fourteen.‡

Neurotic heredity occasionally predisposes to this as to other maladies of the same class. In some cases there is a family history of epilepsy or of insanity, and occasionally of diseases more closely allied to torticollis. The brother of one patient had suffered from facial spasm, and the sister of another (who was suffering from the true, not the hysterical form) had suffered from torticollis itself, and had recovered. In some instances the patient has himself suffered, at a previous time, from some other functional disorder of the nervous system, migraine, &c. In one case of persistent torticollis, the patient, ten years previously, had been affected with general tremor, which lasted for three years and then passed away. The affection has followed an attack of melancholia.§ It is possible that true torticollis may grow out of a habit-spasm also; one patient had been liable, for twenty years, to nodding movements of the head when he was engaged in earnest conversation.‖ In another case there had been for a year occasional half-voluntary movements of the head, in the direction in which it was afterwards moved persistently, by involuntary spasm, while in a third true torticollis was distinctly produced by intentional turning of the head to look at a certain object, continued for several years. Depressing emotion often precedes the onset, but less frequently than in the case of facial spasm. In other instances there is a history of some feebleness of general health. I have more than once known the affection to follow a fall at an interval of a month or two, and a similar sequence has been recorded by others; in one recorded case, spasm on the right side of the neck followed a fall on the left temple.* In most of the cases of apparently traumatic origin, however, the affection followed a fall or other injury in which the neck was forcibly bent or twisted. In some cases the disease seems to be distinctly excited by exposure to cold. Thus one patient had a severe cold, and a rheumatic stiff neck; as the stiffness passed off, clonic spasm came on. But an initial feeling of stiffness is sometimes taken as evidence of exposure to cold, without any real grounds for the assumption. It is remarkable how seldom any peripheral irritation or pain can be traced as the cause of the spasm. It has, however, been observed to follow inflammation of the middle ear; and in the case mentioned above, in which movements like habit-spasm preceded the torticollis, the onset occurred during an attack of severe neuralgia of the fifth nerve, which lasted for three months. The rarity of this cause may be due to the fact that hardly any form of pain about the head or neck is lessened by rotation of the head, or induces such rotation for its relief. Excessive use of the muscles is probably an occasional cause.† It is possible that, in rare cases, spasm in the neck is caused by malarial poison.‡ In not a few cases no immediate cause can be traced; the affection comes on in robust individuals, who are apparently in perfect health at the time of the onset.

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* In the only earlier case that I have seen, in which the spasm commenced at thirteen, and still existed at twenty-nine, the movement was somewhat unusual in its character, and was probably a persistent habit-spasm, from another form of which the patient had also suffered. The rare infantile cases are not included.

† See note on p. 662. It is important to remember that most varied and obstinate forms of clonic spasm occur in the subjects of hysteria, and are apparently in some way due to this disease. They are described in the chapter on hysteria.

‡ Southam, 'Lancet,' Aug., 1881.

§ Allen Sturge, 'Lancet,' 1879, i, 160.

‖ Reynolds states that he has known the disease to follow "writers' cramp" ('System of Med.,' vol. ii, p. 787); but it is probable that it was merely the com-

Among the causes of torticollis commonly described is paralysis of the muscles of one side, permitting their opponents to draw the head to the other side. This is a theoretical cause, which has little counterpart in fact. It is doubtful whether a true paralytic torticollis is ever seen. The amount of muscular action that is needed to keep the head

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mencing spasm (beginning, as it sometimes does, in the hand) that interfered with writing. This was so in one case under my own observation.

* T. Fournier, "Le Tic rotatoire," 'Thèse de Strasbourg,' 1870.

† Annandale has recorded a case of spasm in the neck apparently due to this cause. The subject was a girl aged twenty-four, who, in the course of her work as a weaver, had occasion to move the head rapidly first to one side and then to the other, but especially to the left, and in the spasm the head was inclined and rotated to the left side. But the case was very unusual in that there was no spasm when the head was turned to the left, although clonic spasm came on at once when it was moved out of that posture ('Lancet,' 1879, i, p. 555).

‡ As in a case of intermittent contractions of one sterno-mastoid (coming on each day at the same time, and lasting four or five hours) in a child of four, recorded by Simon. The child had had intermittent fever, and the attacks were quickly cured by quinine (ref. in 'Lancet,' 1879, i, 26).

at rest in the mid-position is extremely small; the rotators are numerous, and all are never paralysed.

SYMPTOMS.—The spasm itself is usually the first symptom, but in a few cases this is preceded or accompanied by pain, or by some unpleasant sensation. The pain may be of an acute neuralgic character, or it may be a dull aching pain. It may be felt in the neck, or in the occipital region on one side, or behind the ear, or in the arm. In other cases, again, a sense of stiffness in the neck attends the commencing spasm. Less commonly the onset is preceded by sensations of a more or less vertiginous character, which may perhaps indicate some morbid action in the nerve-centres, of which the spasm is the result. Thus in one case the spasm commenced a few weeks after a sudden, brief, "whirling" sensation in the head. Another patient at first, when walking, experienced a strong tendency to deviate to one side, towards which there was a slight rotation of the head, but as the rotation increased to distinct spasm, the tendency to deviate ceased.

The actual onset of the spasm is usually gradual, and some months pass before a considerable degree is attained. It may be occasional only, even for years, chiefly during excitement. Very rarely it comes on acutely, in the course of a single week, or even in a single day. Once developed, its increase is usually steady, but now and then it ceases after a few months, and recommences before many more months have passed. Occasionally the interval is longer, even years. In one case, of a middle-aged woman, it ceased after a year's duration, and returned at the end of two years in persistent form. In the case mentioned above, in which the spasm commenced during acute neuralgia of the fifth nerve, it was at first tonic, and reached a high degree of severity in three months, but passed away at the end of six months from the onset, remained absent for eight years and then returned, not again to cease. The tendency of the head to go to one side is often first felt when the patient is walking or excited, and ceases during rest or mental tranquillity. The seat of the first spasm is generally that in which the contractions are ultimately most severe; in exceptional cases it commences in some other part. In two cases it began in the hand, and in one in the face, although in each the neck was afterwards chiefly affected.

The symptoms vary according to the muscles that contract most strongly. These are generally the large muscles of the neck, and in the most common form, torticollis proper, the spasm is either confined to one side, or is greater on one side than on the other, and hence its effect is to cause a deviation of the head to one side, either a rotation or a lateral inclination. Less commonly, as already mentioned, the muscles of both sides are equally involved, and the head is then inclined backwards. Usually many muscles are affected; in some cases one only. The sterno-mastoid is the muscle in which the spasm is most frequent, and in the cases of solitary spasm, it is almost always this muscle that contracts. Of thirty cases, there was some spasm in the sterno-mastoid, on one side or on both, in no less than twenty-seven. In two thirds of the cases (nineteen) the muscle was affected on one side only, and in seven no other was involved. The muscle most frequently associated with the sterno-mastoid is the upper part of the trapezius, and it is generally the trapezius on the same side. Of seven examples of this combination, the associated trapezius was on the same side in six, on the other side in one. But the splenius is associated with the sterno-mastoid almost as frequently as the trapezius, and it is usually the muscle of the opposite side. The scaleni occasionally contract; I have once known both scaleni to be involved with one sterno-mastoid. Often there is also spasm in the platysma myoides, which draws up the skin into characteristic folds, but does not influence the position of the head. In rare cases, of which I have only seen one, rotation of the head is produced by the deeper muscles alone, probably the recti and obliqui, and no accessible muscle can be felt to contract.

The side on which the muscles contract most strongly is more frequently that on which the muscles turn the head to the left, and hence this is the common direction towards which the head deviates. Thus the right sterno-mastoid was involved most or only, rather more frequently than the left. The right trapezius, which also turns the head to the left, was likewise affected more frequently than the left, and the left splenius, which turns the head to its own side, was involved far more frequently than the right. Although the cases are not numerous, the frequency with which this rule obtained can scarcely be a matter of accident. It might be anticipated, *a priori*, that the muscles which move the head to the right would suffer most, because they are associated in action with the right arm; but it must be remembered that many energetic movements of the right arm are effected towards the left.

In the cases in which the muscles of the two sides are equally involved, the sterno-mastoids are also most frequently affected, but usually in slight degree. The trapezii and sometimes the splenii contract strongly. Rarely all the muscles of the neck seem to be in action.

The movement of the head caused by the spasm necessarily differs according to the muscles that are chiefly implicated. We have seen that the spasm is seldom limited to a single muscle; more often several muscles contract together, and the movement of the head is that due to their combined action. As the combinations are numerous, and the muscles contract in various degrees, the resulting movement of the head varies considerably in different cases, and sometimes in the same case at different stages of the disease.

The *sterno-mastoid*, acting alone, causes rotation of the head so as to bring the mastoid process nearer to the inner end of the clavicle.

In this movement the face is turned towards the opposite side, the chin is put forwards, and the head is slightly inclined towards the side of the acting muscle (Fig. 152).

The highest part of the *trapezius* attached to the skull, when acting alone, slightly rotates the head towards the other side, but it inclines the head strongly towards its own side and at the same time draws the head backwards. It also raises the shoulder, rotating the scapula. The middle and lower parts of the trapezius are seldom involved.

The *splenius* of one side inclines the head, and very slightly rotates the face towards the same side.

Equal contraction of the trapezii and splenii, on both sides, causes a backward movement of the head.

FIG. 152.

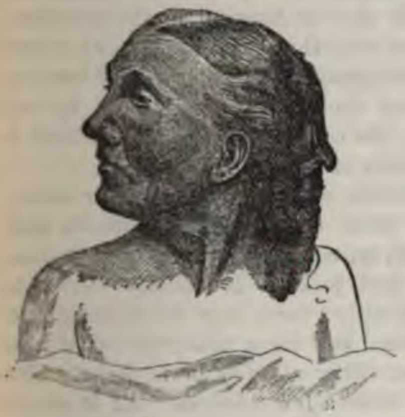

FIG. 153.

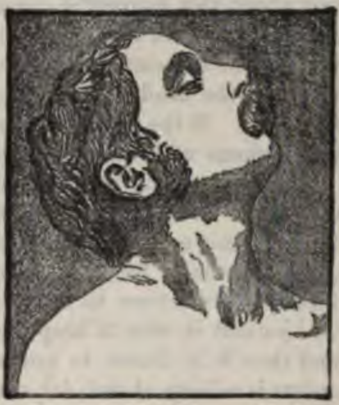

FIG. 152.—Torticollis due to spasm in the left sterno-mastoid.

FIG. 153.—Torticollis. Spasm, chiefly tonic, in the right sterno-mastoid, and especially in the right splenius, with slighter spasm in the left splenius. The patient was a man, aged twenty-six, in whom the spasm had existed for six months; it commenced gradually after a period of great mental anxiety.

The effects of the chief combinations are as follows:—The association of the sterno-mastoid and the trapezius of the same side greatly increases the inclination of the head towards the shoulder, and this increase in the inclination prevents any increase in rotation. The association of the sterno-mastoid of one side with the trapezius of the opposite side prevents the inclination of the head, and does not appreciably lessen the rotation caused by the former muscle, the trapezius being a feeble rotator. Hence the rotation of the head is often very great. The combination of one sterno-mastoid and the opposite splenius causes extreme rotation of the head, so that the face, as in Fig. 152, may be turned towards the shoulder. I have not seen any case in which one sterno-mastoid was associated with the splenius of the same side, but the resulting movement would probably be similar to that produced by the combination with the trapezius of the same side, a strong inclination of the head towards the shoulder. In the case shown in Fig. 153 both splenius muscles were involved (the right most), as well as the right sterno-mastoid, and a strongly backward inclination, with some rotation, was the result.

Bilateral spasm, equal on the two sides, always causes a backward movement of the head, because both sterno-mastoids scarcely ever contract alone, and the spasm in the muscles at the back of the neck draws the head backwards, and then the inclination is increased by the sterno-mastoids. It is doubtful whether there is ever a forward movement in any case properly belonging to the group now under consideration. The retroflexion of the neck is sometimes very great; the face may even be horizontal, turned directly upwards, as in one case that I have seen. Such extreme spasm is, however, rare; more often the contraction is moderate in degree. An interesting fact about this retrocollic spasm is that contraction in the frontales muscles is almost always associated with the spasm in the back of the neck. The association is a physiological one; in the act of looking upwards the head is bent back, and the eyebrows are raised by the frontales. If the spasm is clonic, the contraction in the forehead is synchronous with that in the muscles of the back of the neck.

The spasm in all forms of torticollis may be either tonic or clonic, and often both kinds of spasm occur together; clonic spasm may become tonic as it becomes intense, or, more commonly, tonic spasm, as it increases, becomes clonic and jerky. It usually varies in intensity from time to time, and either form may be intermittent. The patient is able to keep the head still for a time, but every now and then it is drawn to one side, either steadily or in jerks. The spasm is seldom absent for more than a few minutes. As it comes on, the affected muscles stand out conspicuously in their contraction. Occasionally the spasm, either tonic or clonic, is practically continuous. The same variations are seen in the retrocollic variety, but clonic spasm is relatively more common, and intermissions are less common. The contractions are often moderate in degree and frequent in recurrence, so that the spasm (especially at first) bears more resemblance to tremor than in the ordinary forms.

In most cases there is a strong tendency, in the course of time, for the spasm to spread and to involve other muscles in addition to that in which it commenced. Even when the contractions are limited, if the movement of the head is forcibly prevented, the spasm often temporarily spreads to other muscles. Thus, in a case in which one splenius alone habitually contracted, if the movement was prevented, the sterno-mastoid began to contract.

In some cases the spasm is not limited to the muscles of the neck; it involves either the arm, or the face, or the muscles of mastication. The face is seldom constantly involved, but muscles occasionally contract during a severe paroxysm. In one instance, with clonic spasm in one sterno-mastoid and trapezius, at the height of the attack, there was often twitching of the eyelids and pouting of the lips. The spasm in the face may occur on the side on which the sterno-mastoid contracts, or contracts most strongly. In one instance, in which there was continuous clonic spasm in the right sterno-mastoid, and very little in the left, there were almost constant contractions in the right side of the face. Rarely the spasm is equal on the two sides of the face when the spasm in the neck is unequal, as in a case just mentioned. In retrocollic spasm there may be some bilateral contraction in the lower facial muscles, especially in the elevators of the upper lip, at the height of a paroxysm, in addition to the constant spasm in the frontales, already described. The masseters are involved in true torticollic spasm rarely, and only at the height of a paroxysm. In chronic retrocollic spasm they are sometimes constantly affected.

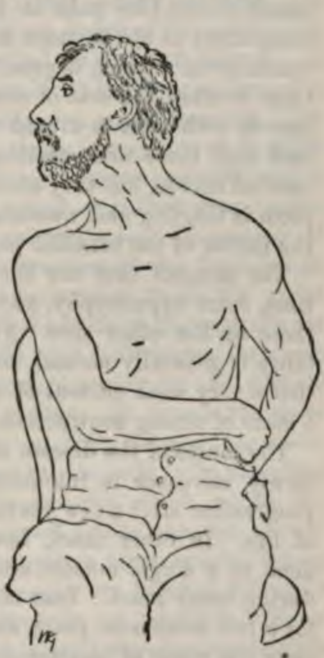

FIG. 154.—Spasm, chiefly tonic, in the left sterno-mastoid, right trapezius, and the whole of the right arm, which was strongly adducted by spasm in the pectoralis. The patient was a man aged forty-six, in whom the spasm had existed for two years. It commenced gradually in the hand.

When the arm is involved, there is always unequal spasm in the two sides of the neck. The sterno-mastoid that is most affected may be on the same side as the arm, or on the opposite side. In the cases that I have seen in which the arm was affected after the neck, it was the arm on the side opposite to the most affected sterno-mastoid. When the arm is affected first, either sterno-mastoid may be the seat of the chief spasm, and the head may thus be turned from, or inclined towards, the arm that is affected. In one case of clonic spasm in the right sterno-mastoid, there was also spasm in the left deltoid, but in no other muscle. Another instance of this relation is presented by the patient shown in Fig. 154. In him the spasm commenced in the arm and spread to the neck, where it involved the right sterno-mastoid, the left trapezius and splenius, and the right platysma. It became continuous in both arm and neck, so that the upper arm was in constant strong adduction, and the forearm was carried in front of the body, while the face was turned towards the shoulder.

As an instance of the peculiar forms met with occasionally in infants may be mentioned that of a girl, aged eleven months, whose head was drawn strongly to the left, and agitated by clonic spasm for a few minutes, twelve to twenty times a day. The affection had commenced acutely, without obvious cause. At first each attack was attended by deviation of the left eye to the right, and by nystagmus in this eye during the intervals, but the eye symptoms ceased after a few weeks. A sister of the child was said to have suffered in the same way, for a time, at about the same age.

Considerable spasm always occasions discomfort to the patient, partly due to fatigue in the muscles. There is not commonly any actual cramp-like pain in them. The neuralgic pain that is often complained of at the onset usually ceases after a time, but sometimes continues in varying degree. When there is spasm in the arm, the limb is often the seat of considerable pain, and I have known severe pain in both arms to attend the attacks of spasm in the neck. It is said that there are occasionally tender points in the course of the cervical nerves, but they are not common. Another rare sensory symptom is tingling and numbness in the arm, from the compression of the nerves of the brachial plexus by the scaleni.

The muscles that are the seat of considerable spasm undergo, in time, some hypertrophy, and may become very distinctly larger than those on the other side. They never waste. The electrical irritability is generally normal, sometimes indeed it is abnormally great, so that a very weak current of either kind suffices to put the muscles in a state of strong contraction.

The course of the disease varies in different cases. Often the spasm slowly increases in intensity and widens in range, but ceases to be progressive after a few years, and may remain stationary for the rest of life. In other cases, however, it remains slight, sometimes confined to a single muscle, and may not undergo perceptible increase during many years. Less commonly it diminishes after it has lasted for a few months or years, and may pass away, either spontaneously or as the result of treatment, and it may return after a variable period of freedom, as in the cases mentioned in the section on causation. In some instances it does not disappear altogether, although it becomes slight and occasional. In a case recorded by Brodie, the spasm ceased, for the time, during an attack of insanity, and I have seen an analogous case. The disease does not develop to any disorder of more serious character, nor has it any tendency to shorten life, although it may lessen very much the pleasure of existence.

PATHOLOGY.—In no case of torticollis has a lesion been found that can be regarded as an indication of the morbid process to which the spasm is due. The pathology of the disease is therefore a matter of inference from its symptoms, its causes, and from what we know of the nature of allied diseases, especially of facial spasm (see p. 248). It is almost superfluous to state that the morbid process cannot be in the muscles themselves. The facts that many muscles are involved, and that when the spasm commences in a single muscle it usually spreads to others, as well as the general pathology of spasm, make it probable that the muscular contractions depend on the over-action of nerve-cells, and not on any irritation of nerve-fibres. But we do not know what nerve-cells are primarily deranged, whether they are the cells of the lower spinal centres, or those of the higher cortical centres. The symptoms suggest that in some cases the lower, and in other cases the higher centre is that from which the discharge originates, because the distribution of the spasm indicates that the functional level, so to speak, of the discharge is not always the same. In some, the affected muscles are those that act together in producing a given movement, although they are supplied by different nerves. It seems probable that such a spasm depends on the over-action of a centre in which movements, rather than muscles, are represented. On the other hand, when the spasm is of a single muscle, or of muscles that are not physiologically associated, it seems more probable that it depends on a lower centre, such as the spinal or bulbar grey matter from which the nerves proceed. The physiological association of muscles is specially noticeable in the contraction of the frontales in retrocollic spasm. These muscles are innervated from the facial nucleus in the pons, while the muscles at the back of the neck are for the most part innervated from the spinal cord. It seems far more probable that such an associated spasm depends on the cortical centres than that it is due to the lower centres. So, too, in the cases in which there is spasm in the muscles, on both sides of the neck, that are concerned in turning the head to one side. On the other hand, such an association as that of the muscles which incline the head towards one shoulder, or strong spasm limited to one sterno-mastoid and the opposite deltoid muscles, seems more intelligible on the assumption that the over-acting nerve-cells are those of the lower centres, since these muscles do not commonly act together without others. The causation of the disease by injuries that involve forcible bending of the neck also suggests that, in such cases, the spasm proceeds from the lower centre. We have seen reason to believe that the over-action that causes facial spasm is sometimes in the cortical centres, and sometimes in the lower centres. At the same time we must not lay too much weight on the significance of functional association. It is probable that the connection of the cells of the lower centres is determined by this association, and the manner in which the spasm spreads among the muscles on both sides is often more intelligible on the assumption that the over-acting cells are those of lower centres. It may be noted, moreover, that there is reason to believe that structures in the lower part of the pons Varolii influence the associated action of rotation of the head (see p. 187). There is one important difference between the spasm in torticollis and that which occurs in the same muscles as a consequence of cortical disease. In the latter a conjugate deviation of the eyes is usually associated with that of the head, and this is true also of the spasm that results from irritation of the structures in the pons. In ordinary torticollis, however widely the spasm spreads, the muscles of the eyeball are not implicated. From these considerations it seems doubtful whether we have at present sufficient evidence to justify us in coming to any conclusion regarding the seat of the primary over-action of the cells which causes the symptoms.

The nature of the morbid process is also involved in as much obscurity as it is in all other similar spasmodic diseases. That there is an unnatural state of the nerve-cells, in consequence of which nerve-force is spontaneously liberated, is scarcely more than a statement of the observed facts, and leaves us still ignorant of the condition on which the phenomena depend. There is at present no evidence that the over-action of the cells depends on any morbid process outside them, or any lesions that could be detected by naked-eye or microscopical examination. The fact that neurotic heredity can often be traced as a predisposing cause, suggests that the over-action has its origin in a primary derangement of the nerve-elements themselves. It is to be noted that this disease, in common with other spasmodic affections of the same class, comes on usually in adult life, and seems to depend on a local failure in the stability of function that has been long developed, and cannot therefore be ascribed either to an imperfect establishment of function on the one hand, or to actual senile changes on the other. In their occurrence at the time when nerve-action might be expected to possess its full stability and precision, and also in their local character, these affections constitute an enigma to which we have as yet no solution.

DIAGNOSIS.—The diagnosis of spasmodic torticollis seldom presents any difficulty. The condition that has been termed "false torticollis," in which there is a deviation of the head from some other cause than muscular contraction, is readily distinguished from the variety that is due to shortening of one sterno-mastoid (with which alone it is likely to be confounded) by noting the relation between the position of the head and the side on which the muscle is tense. In the spurious form the sterno-mastoid is tense on the side towards which the face is turned; in the true form the tension is of the opposite muscle. Retrocollic spasm, when the movements are small in range, is apt to be mistaken for simple tremor, but the muscular contractions can be felt to be more violent than they ever are in simple tremor, and the associated spasm in the frontales, which has been conspicuous in all the cases I have seen, supplies an absolute distinction between the two affections. Another occasional difficulty is the distinction of true torticollis from hysterical spasm of torticollic type. The facts of etiology show that under thirty true torticollis seldom occurs in females, and therefore at that period there will always be a presumption that the affection is hysterical, but after thirty the mere fact of sex must not be allowed weight. As a rule, hysterical spasm tends to spread from the neck to the trunk, which becomes affected by writhing movements. In all the cases that I have seen in which the conditions of onset raised a suspicion of the hysterical nature of the case, and the spasm was limited to the neck, and was typical in form, the affection turned out to be the true variety. There is, indeed, no absolute criterion by which this point in diagnosis may be determined. I have even known recovery, under treatment suited for an hysterical affection, to be followed by a return of persistent spasm.

PROGNOSIS.—The facts of the course of torticollis, already mentioned, show that the prognosis must be grave in every developed case. The more severe the spasm, the wider its extent, and the longer its duration, the smaller is the prospect that considerable relief or cessation will be obtained. This is, however, greater in the first half of adult life than in the second, and it is better when the spasm is variable than when it is uniform in seat.

TREATMENT.—The removal of any influences that can be regarded as having helped to induce the disease is, of course, of the first importance. Cases do not often come under treatment in the early stage, but it is desirable that any stiffness due to cold, that has lasted for an unusually long time, should be treated by rest and hot applications. Unfortunately it is very rare for causal treatment to have any appreciable influence on the disorder. The same may be said of tonic treatment in general, necessary as it is to give tonics whenever they are indicated. Drugs that may be called "stimulant nervine tonics," such as valerianate of zinc and asafœtida, sometimes have a marked influence on the spasm, even in cases in which it is certainly not hysterical in nature. In one, the adminstration of these two drugs, without other treatment, reduced severe spasm to a very trifling degree of intensity, and the improvement continued for some months, but the spasm then increased again and resisted all remedies. Sedatives frequently lessen the spasm while their action on the system continues, especially opium, morphia, chloral, succus conii in large doses (gradually increased to two ounces), bromide of potassium, and Indian hemp. But the discontinuance of the drug is usually followed by a relapse to the former degree of severity, although occasionally this does not take place for some weeks or even months. I have only once known great and permanent improvement to result from the internal administration of sedatives, which in this case consisted of the combination of bromide of potassium and cannabis indica. There is one mode of treatment that has in many cases had a more lasting influence, but it is a remedy that has to be carefully weighed in the balance against the disease,—the hypodermic injection of morphia. Continued for several months, in doses increased gradually to half a grain or a grain a day, it has entirely removed the spasm. But the patients so treated find the discontinuance of the drug extremely difficult.

not only on account of the craving for it that is established, but also because (what is perhaps part of the craving) they feel, or imagine they feel, a tendency to the return of the spasm if the drug is discontinued. In one very severe and distressing case of retrocollic spasm, in a man aged forty-five, all muscular contractions ceased under the influence of the drug, and the patient has now been free for ten years, but has never left off the use of morphia, although he has not increased the dose above that named. A patient so treated should undergo a subsequent course for the eradication of the morphia habit, before he passes from the hands of the practitioner, and the effect of the withdrawal of the morphia should be carefully observed. Cocaine may be used, but for a time only, as the morphia is being withdrawn. Unless these measures are adopted, the treatment is the substitution of one morbid state for another, and that which is substituted may be in the end the more harmful of the two. The patient above mentioned, however, has had ten years of perfect comfort in exchange for a condition that was most distressing; the spasm was so severe that he could never enter a public conveyance on account of the painful observation he excited. Reynolds has also described favorable results from the treatment.* The addition of arsenic to the injected morphia has been advocated by Radcliffe, but arsenic alone, either by the mouth or by hypodermic injection, seldom has much influence,† and it is therefore probable that the influence of the combination is due chiefly to the morphia. The beneficial influence of internal remedies has been chiefly observed in cases in the first half of adult life, in which there is occasionally a distinct tendency for the spasm to subside and even cease. The energetic and prolonged use of morphia is, however, an exception to this, since it has been equally effective in later life.

Electricity has been used in various ways in the treatment of the disease. The method that has generally been employed is the application of the voltaic current to over-acting muscles, a weak current being used, which has some sedative influence. The positive pole may be placed just below the occiput or on the highest accessible part of the nerve, and the negative on each contracting muscle for five or ten minutes. Thus employed, it has been said to do good in some cases; in one of severe bilateral spasm, recorded by Poore, the affection almost ceased under the treatment, but the case was peculiar, and possibly allied to the hysterical form. In general, electricity fails to do more than produce a slight and transient diminution in the spasm; I have not myself seen any permanent good result from the agent, even when long continued. Faradisation of the antagonists of the over-acting muscles has been recommended, but this method is as destitute of rational foundation as it certainly is of practical effect. The disease probably never results from the weakness of the opponents, and cannot be lessened by increasing their activity. A blister over the contracting muscles may lessen the spasm for a time in very marked degree, but the effect is seldom as permanent as it often is in hysterical spasm. Blistering over the cervical spine, however, apparently arrested the affection in one instance.*

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* 'System of Med.,' vol. ii, p. 797.

† A case of clonic spasm which recovered under the administration of arsenic, recorded by Buzzard, was certainly of hysterical nature ('Brit. Med. Journ.,' 1881, p. 937).

Mechanical supports afford occasionally some relief, which depends on the fact that the greater the movement and muscular contraction, the greater is the discomfort produced. Many patients are comparatively easy when they are sitting in a chair with a high back, against which they can fix the head. The best support is one in which a rigid rod is carried up to the back of the head, and from its top a small pad projects on the side towards which the occiput is turned by the spasm, so that the patient is able to press the head against the pad. The instrument needs careful contrivance, having to be specially adapted to each individual case, and it is only when the spasm is moderate in degree that the expedient is very successful. Instruments that are designed to fix the head rigidly, and prevent all movement, can never be endured.

Surgical measures have been frequently employed in the treatment of torticollis, but, with one exception, their effect has been slight and disappointing. The most useless procedure that has been employed is the division of the tendon of the contracting muscle. This has been sometimes done apparently because the operation cures the totally different fixed wry-neck. In active spasm it is worse than useless, because the division of the tendon permits the muscle to shorten, while it does not check the spasm, and the greater the contraction of the muscular fibres the greater is the pain occasioned by the contraction. In one case that came under my notice some time after the operation had been performed, the patient's suffering was much increased, and, possibly in consequence of the increased pain, the spasm had spread to other muscles.

In many cases in which the spasm was confined to, or greatest in, the sterno-mastoid, or in this and the trapezius, the spinal accessory nerve has been stretched. Temporary relief has followed, but when the effect of the stretching had passed away, the spasm has almost invariably returned in its original severity.† The only operation that can be credited with an approximate or actual cure of the affection is division of the nerve, with excision of a piece, half an inch or so in length, to prevent reunion of the ends. The operation causes, of course, permanent paralysis and atrophy of the muscular fibres to which the nerve is distributed, of the whole muscle if, as in the case of the sterno-mastoid and upper part of the trapezius, there is no other nerve-supply. All spasm in the muscle thus paralysed is necessarily abolished. Campbell de Morgan first cured a patient (in 1862) by this procedure, and it has since been employed with success in many cases. When other muscles have also been involved, in slighter degree, the arrest of the spasm in the muscles first and most affected has frequently been followed by a remarkable diminution in the contractions in the other muscles, which have subsided to a merely trifling degree, and sometimes have gradually ceased. When other muscles, as the splenius, complexus, &c., have been the seat of severe spasm, this has commonly persisted after the operation on the spinal accessory nerve; but it has been shown by Keen and Noble Smith that this spasm may also be reduced to a slight degree, or removed, by the subsequent division of the posterior branches of two or three of the cervical nerves supplying the muscles involved. Neurectomy is certainly the most successful method yet employed in the treatment of torticollis. The paralysing effect of the division of the branches of the spinal nerves is unimportant; the palsy of the sterno-mastoid interferes with the movement of the head in some degree, but very little with its position; that of the trapezius lessens the power of the arm, but this is a far slighter inconvenience to the patient than the spasm which it replaces. The remarkable subsidence of moderate spasm in other muscles than those chiefly affected, may perhaps be due to the fact that the operation necessarily arrests the transmission, not only of the motor impulses to the muscle, but also of the afferent impulses from the muscle to the centre. These, generated by the muscular contractions, must be intense and constant, and may be concerned in the extension of the central over-action.[*]

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* Morton Prince, 'Boston Med. and Surg. Journ.,' 1886.

† The only case in which great improvement followed stretching was very unusual in the fact that the patient was a boy of fourteen, and the spasm affected also the muscles of the back and both arms. It was probably more closely allied to the hysterical than to the ordinary form. (Southam, 'Lancet,' 1881, ii, p. 369.)

TETANUS.

Tetanus is a disease of the nervous system, characterised by persistent tonic spasm, with violent brief exacerbations. The spasm almost always commences in the muscles of the neck and jaw, causing closure of the jaws (*trismus, lockjaw*[†]), and involves the muscles of the trunk more than those of the limbs. It is always acute in onset, and, of those who are attacked, a very large proportion die. The disease is usually the result of a wound (*traumatic tetanus*), but

[*] Among the cases of successful neurectomy described and discussed are those of De Morgan, 'Med.-Chir. Rev.,' 1866, and 'Lancet,' Aug., 1867; Annandale, 'Lancet,' 1879; Sands, Tillaux, and Hansen; cases quoted by Bowlby, 'Injuries and Diseases of Nerves;' Ballance, 'St. Thomas's Hosp. Rep.,' 1884; Keen, 'Annals of Surgery,' Jan., 1891; Noble Smith, 'Brit. Med. Journal,' 1891, and a pamphlet, 'Spasmodic Wry-neck,' in which are quoted the details of all the published cases.

[†] Properly "locked jaw."

sometimes occurs without external injury, especially after exposure to cold (*idiopathic* or *rheumatic tetanus*). It occurs also in newly-born children (*tetanus neonatorum, trismus nascentium*), and, rarely, after childbirth or abortion (*puerperal tetanus*).

Etiology.—Traumatic tetanus is far more common in males than in females, the proportion being nearly as 6 to 1.* The idiopathic form also occurs in males more frequently than in females, although the disproportion is not quite so great as in the traumatic form. Of 46 idiopathic cases 37 were males and 9 females, a proportion of 4 to 1. Doubtless the chief cause of the different liability of the sexes is the greater exposure to the immediate causes, involved in the occupations of men.

Tetanus occurs at every period of life. The distribution of 160 traumatic cases and of 46 idiopathic cases is shown in the tables below. During the first five years of life (the first month excepted) there is almost complete immunity, the cases under ten occurring after five. The second decade of life yields the largest proportion, rather more than a quarter; the third and fourth decades, each rather less than a quarter, so that, in the thirty years of life from ten to forty, about three quarters of the total number of cases occur. The disease continues, with decreasing frequency, up to old age, and has been met with as late as eighty-nine (Yandell). The few cases in childhood (five to ten years) occur in each sex with almost equal frequency; from ten to forty the proportion between the sexes is near the average (1 to 6); between forty and fifty, women suffer still less frequently, and scarcely ever after fifty.

TRAUMATIC	1–9	10–19	20–29	30–39	40–49	50–59	60+
Males	5	36	32	29	20	11	5
Females	4	7	4	5	2	0	0
Total	9	43	36	34	22	11	5
Per cent.	5·5	27	22·5	21	14	7	3

IDIOPATHIC	1–9	10–19	20–29	30–39	40–49	50–59	60+
Males	2	5	9	12	4	0	3
Females	1	4	0	2	2	0	0
Total	3	9	9	14	6	0	3

Dark-skinned races have been observed to suffer from all forms of tetanus more frequently than Europeans when both are exposed to the same influences. In hot countries, *e. g.* India and the West

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* The published series of cases at Glasgow (Lawrie, 'Glasgow Med. Journal,' 1853, vol. i, p. 339), and at Guy's Hospital (Poland, 'Guy's Hosp. Rep.,' 1857; F. Taylor, 'Guy's Hosp. Rep.,' 1878), idiopathic cases excluded, comprise 160, of which 138 were in males and 22 in females. Consecutive series of cases such as these give more accurate statistics than collections of cases which have been published separately (often on account of some special feature), and also than the more massive but undifferentiated statistics of the general mortality.

Indies, tetanus is far more common than in temperate regions, and it is especially prevalent at certain periods and in certain places. In temperate climates, however, its incidence does not appear to be influenced by weather or by season.

It is doubtful whether previous health exerts an influence on the occurrence of the disease. The robust suffer as well as the weakly. It has been thought that depressing emotions predispose to it, and especially a fear of the disease, but it is doubtful whether this is really proved by ascertained facts.*

The *immediate cause* of tetanus is a specific bacillus, which, in the usual traumatic form, is inoculated at the wound. The symptoms depend, however, not directly on the organism, but on a toxic material produced by it, which seems to have an action on the central nervous system analogous to that of strychnine. The evidence of this causation is conclusive; it has been ascertained by the researches of numerous investigators during the last six years, and will be described in the section on Pathology. The bacillus exists chiefly in earth-mould, especially where any putrefaction is going on, as in manured soil; but it is widely spread, and its spores are probably carried also by the air. The discovery explains many facts of causation that were before mysterious, as, for instance, the prevalence in certain localities; while the fact that the bacillus thrives best at a comparatively high temperature explains the frequency of the disease in hot countries. But the precise relation of other causal conditions to the action of the organism has still to be cleared up. The discovery reduces to a subordinate position the influences which were formerly regarded as the chief causes of the disease; they must not, however, be ignored, although they need fresh study in the light of the new facts. For instance, the different susceptibility of the dark and light-skinned races indicates a difference in the liability of the individuals, analogous to that which, as we shall see, exists among different animals, and it suggests that variations in susceptibility may exist in the same individual, at different times, as the result of influences which formerly were alone discerned.

In traumatic tetanus the wound involves an actual breach of the surface; it may be in any position, of any character, and of any degree of severity. Cases have been recorded in which tetanus has followed the most trifling injuries (the stings of bees and wasps, the prick of a thorn, the removal† of a foreign body from the eye), every kind of incised, punctured, contused, and lacerated wounds, compound fractures, and almost every form of surgical operation, from the extraction of a tooth to ovariotomy. But it is far more common after punctured, contused, and lacerated wounds than after incised wounds. Hence it is comparatively rare after surgical operations. Injuries of nerves have been supposed to be especially concerned in its production, and those of small nerves rather than those of large nerve-trunks. It also follows extensive burns, and also severe frostbites. The order of frequency* with which the several parts have been the seat of the wound that caused tetanus is (1) hand, (2) leg, (3) foot, (4) head and neck, (5) arm, (6) trunk. The wound has sometimes been in an unhealthy, irritated condition; more frequently it has been in a perfectly healthy state, and sometimes cicatrisation has been advanced, or even complete. Since attention has been given to the point, it has been observed that in a considerable proportion of the cases the injury involved contamination of the wound with soil, as in falls on the ground, a puncture by a broken stick or stake which had been in the earth, or by a splinter from a dirty floor. Such a splinter from the floor of a skittle alley, penetrating beneath the nail, produced it; one fatal case was due to a compound fracture of both femora, from a fall in which the ends of the bones were covered with earth; gardeners have suffered from punctures by sticks. In most of these cases the tetanus bacilli were found in the source of the contaminating material; their presence explains the influence of these injuries. They have been found in spiders' webs, and tetanus has followed the application of such webs as a styptic (a popular custom in some places), and also the application of earth to a wound. Spontaneous disease—ulcers, for instance—may also occasion tetanus, although far less frequently than wounds. In such cases, and also in burns, the organisms must reach the local lesion either by contaminated applications (possible in the case of ulcers, but unlikely in that of burns), or else through the air. In rare instances the disease is said to have followed injuries which involved no breach of surface, as flogging, and falls on the back of the head and spine. Such cases ought perhaps to be provisionally classed with the idiopathic form, in respect to the mystery of their direct causation.

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* A curious case (described in the last edition), in which dread of the disease apparently excited the malady, has been deprived of its significance by the recent discoveries, since the injury was a lacerated wound of the hand, produced by its contact with the road in a fall from a horse.

† Probably rather its presence there.

The interval between the infliction of the wound and the appearance of the first symptoms of tetanus is usually from five to fourteen days (in two thirds of the cases), but the malady may set in earlier or later. Many cases have been recorded which commenced within forty-eight hours of the injury, a few within twelve hours, and one or two in which tetanus came on almost immediately. In Robinson's often-quoted case, a negro, who cut his hand with a piece of porcelain, was dead in fifteen minuites. On the other hand, tetanus occasionally commences during the third or fourth week after the injury; scarcely ever later than a month.

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* According to 395 cases tabulated by Thamhayn ('Schmidt's Jahrbucher,' vol. cxii, 1861). This does not show its absolute frequency in wounds of each part; for the determination of this no adequate statistics at present exist. It is said by some to be very seldom caused by wounds of the head and neck. Of 505 cases of tetanus tabulated in the records of the American civil war, only 21 were due to injuries of those parts. Nevertheless one peculiar form of tetanus (cephalic tetanus) seems to be produced chiefly by injury in the region of the fifth nerve (see p. 683).

Idiopathic tetanus usually follows exposure to cold, especially to wet cold when the body is perspiring. In many cases the exposure has been repeated and prolonged. When due to a single exposure, so that the interval could be ascertained, it has rarely exceeded two days, and has frequently been only a few hours. In many cases classed as traumatic tetanus there has been an exposure to cold after the receipt of the wound. This has been thought to be the chief cause of the frequency of tetanus in soldiers wounded in battle. The influence of cold has also been thought to aid the occurrence of puerperal and neonatal tetanus. But the relation of the apparent influence of cold to the infective agent has still to be explained. The exposure to cold may sometimes have been merely coincident with that to opportunities of infection, as, for instance, in soldiers on the march. Some influences may predispose the nervous system to suffer from a minute dose of the poison. The relation of the organisms to idiopathic tetanus has also not yet been ascertained. It has, indeed, been maintained that all the supposed idiopathic cases are the result of some unnoticed trifling surface injury (Verneuil). At present this can scarcely be admitted, but the possibility of such causation may be remembered in connection with the rare cases in which no morbid influence can be traced, and with those that have been observed to follow some apparently inadequate cause, alcoholic intoxication, insolation, and violent emotion, and perhaps also the inflammation of some serous membrane, excited usually by cold. Intestinal worms have probably been merely coincident.

Puerperal tetanus is frequent in hot countries, and is perhaps the most terrible form of the disease. It is said that in Bombay, in three years, 232 women died from this cause. Fortunately it is very rare except in the tropics. Of fifty cases in temperate countries* it occurred in eighteen after abortion; in thirty-two after labour at or near the full time. The cases were spread over the childbearing period of life, the youngest patient being twenty-two, the oldest forty-eight. Those after abortion occurred from the second to the fourth month of pregnancy. In most of the cases there was severe hæmorrhage, and in seven it is expressly stated that the vagina was plugged, a procedure that has been thought to influence the occurrence of the disease. The interval between the abortion and the first symptoms varied from five to thirteen days, and in two thirds of the cases it was from five to ten days; in no case was it less than five. In two, instruments had been used to procure abortion, and metritis had resulted. In only one of these cases was the tetanus preceded by exposure to cold, on the tenth day; the first symptoms occurred on the eleventh.

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* Including sixteen of those collected by Simpson ('Edin. Med. Journ.,' 1854). In several cases it has followed the Cæsarean section, but these are not included.

Of the cases after labour, the particulars of the labour are recorded in twenty-six, and in only three was there no abnormal condition during or after parturition. The most frequent accident was adhesion of the placenta, which existed in nearly half the cases (twelve). Metritis occurred alone in two, and in association with pharyngeal diphtheria in one. In only one case was the forceps used; in one there was placenta prævia, and turning was necessary. In four cases the disease followed a subsequent exposure to cold; and in one it succeeded a secondary hæmorrhage, two weeks after delivery, for which the vagina was plugged. The interval before the appearance of the symptoms (noted in twenty-seven cases) varied more than in the cases after abortion, but in nearly half (twelve cases) it was from five to seven days (inclusive). In one third (nine cases) it came on during the second week.

In three cases, which occurred after fourteen days, a secondary influence could be traced (diphtheria and metritis, cold, secondary hæmorrhage). On the other hand, the interval was once only three days, and once four days, and in one exceptional case (without albuminuria) symptoms of tetanus came on during labour, ceased for three hours after delivery, then recurred with such severity as to cause death in an hour.* In spite of the apparent influence of external causes, it is the general opinion that the disease is related to the presence of decomposing substances in the uterus, to which, in some way, the tetanus bacilli gain access, or in which some toxic material is formed, which has an action on the nervous system similar to that produced by the organism. The inhalation of the spores of the bacillus, and their passage to the decomposing substance of the blood, is possible.

Tetanus neonatorum is fortunately a rare disease in this country, but, like other forms, it is exceedingly common in certain tropical countries, especially among the coloured races. In Demerara, at one period, one half of the negro children died from this cause. But it is endemic also in some localities far removed from the tropics, as in the notorious instance of Heimaey, an island near Iceland, where the population at one time was kept up only by immigration, almost all the children dying from this cause. This fact is rendered intelligible by the discovery that the specific bacillus, infecting the umbilical wound, is the cause of this form also. Its presence is doubtless favoured by insanitary conditions, since the employment of well-arranged lying-in hospitals has caused the affection, formerly prevalent, to disappear.† It usually begins between the third and seventh day, occasionally not till the second week, very rarely later.‡

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* Curtis Smith, 'Philadelphia Med. Reports,' 1873.

† Of three successive children of the same mother, the first two, born in one house, died of tetanus; the third, born in a different house, did not suffer (Salzmann, 'Wurt. Cor.-bl.,' 1885). This fact agrees perfectly with the causation of the disease by bacilli. (See Pathology.)

‡ Tetanoid spasms, commencing within twenty four hours of birth, are probably

The liability to tetanus of newly-born children seems to be uninfluenced by sex; boys suffer only a little more frequently than girls.[*] The umbilical wound is frequently inflamed; sometimes arteritis extends from it along the cord,[†] but it may be healthy in appearance. In hot climates (*e. g.* India, according to Wallace) tetanus often results from circumcision, and hence is far more common, during the second and third week of life, among Jews and Mohammedans than among Christians.[‡] Occasionally (as in other traumatic cases) an exposure to cold precedes the onset.

SYMPTOMS.—The symptoms of tetanus present little variation in the majority of cases. Rarely, vague pains in the head, the epigastrium, or, in traumatic cases, at the seat of the wound, have preceded the onset. The first symptom is usually some sense of stiffness on movement of the neck or jaw, sometimes difficulty in swallowing, or stiffness in the tongue. The patient thinks that he has got a stiff neck from sitting in a draught, and if, as is often the case, the symptoms have actually followed an exposure to cold, the patient's impression may be shared by his medical attendant. But in the course of a few hours, or at most of a day or two, the difficulty in separating the jaws becomes greater, and is clearly due to increasing rigidity of the masseters. With this there is also more stiffness in the neck, and the head is slightly bent backwards from the preponderance of the spasm in the extensor muscles. As the rigidity in the neck increases it passes down the spinal muscles, until the vertebral column becomes over-extended in what is called "opisthotonos." The legs may also become extended and rigid, but the arms are little affected. Sometimes opisthotonos comes on at the same time as the rigidity in the muscles of the jaw.

due to meningeal hæmorrhage (see p. 413). Marion Sims maintained that tetanus may be produced by displacement forwards of the occipital bone, irritating the medulla. This displacement, he said, is physiological during birth, and is maintained afterwards, in these cases, by the child lying on its back, with the head resting on the arm of the nurse; the symptoms may be removed by keeping it on its side, first one, then the other, for a few hours ('American Journal of Medical Science,' April, 1846; July and October, 1848). Evidence in favour of this opinion has been brought forward by Wilhite (ibid., 1875, p. 375), and by Hartigan (ibid., Jan., 1884); but these authors are manifestly in error in believing all cases of tetanus neonatorum to be thus produced and thus remediable. Indeed, the correct statement of the facts, if their view is correct, is not that tetanus is thus produced, but that tetanus neonatorum is less common than is supposed, and is simulated by the effects of compression of the brain.

[*] In Ireland, of 371 deaths from tetanus under five years between 1841 and 1851, 219 were of boys and 152 of girls. It is probable that all these cases were of tetanus neonatorum, since, as already stated, the disease is otherwise practically unknown under five years.

[†] Two cases, due apparently to umbilical ulceration, one in a child three weeks old, are described by Godlee and Williams, 'Med. Times and Gaz.,' Dec. 27th, 1884.

[‡] Wallace, 'Lancet,' 1882, Aug. 12th.

As the rigidity increases it involves the facial muscles. Apparently all the facial muscles are affected, but those which are strongest overcome the others, and impress on the countenance the effects of their contraction. Thus the eyebrows are raised by the frontales, while the ocular fissure is narrowed by the orbicularis. The angles of the mouth are drawn outwards and a little downwards, and the upper lip is pressed against the teeth, in what has been termed the "risus sardonicus."*

The tonic spasm or rigidity occasions at first little discomfort beyond a feeling of stiffness, but as it increases a pain is felt in the rigid muscles. Soon, however, it occurs in paroxysms, at first slight, but gradually increasing in severity, and causing distressing cramp-like pain. The paroxysms are usually brief, lasting from five to fifteen seconds, and the spasm is greatest in those muscles which are the seat of the persistent rigidity. Thus the posture produced by the latter is increased during the attacks. The head is drawn back, the spine is arched, the legs rigid, and the feet extended, so that the patient, in a severe paroxysm, may rest on the head and heels. The peculiar expression of the face is exaggerated during the attacks; the jaw is firmly closed, and sometimes the tongue is bitten from being caught between the teeth when the paroxysm comes on. Other muscles, however, are involved, although in less degree. The thorax may be fixed and the glottis closed, while the face becomes livid, in consequence of the interference with respiration. The abdominal muscles are also contracted and hard like boards, while the arms, though free from rigidity at the elbow and hand, may be fixed to the thorax. In addition to the cramp-like pain in the muscles, severe and very distressing pain is frequently felt in the epigastric region, darting through to the back. It may be an early symptom, and is supposed to be due to spasm in the diaphragm. From the violence of the muscular action the surface is covered with sweat, and during the paroxysms perspiration may pour in streams from the body.

Among the variations in the symptoms it may be noted that in rare cases the spasm commences in the limb injured, or movement of the limb may excite a paroxysm in which the spasm commences there and spreads gradually. Occasionally the stiffness in the throat precedes that in the jaw, and may be described as "sore throat." It has been known to continue for a week before the rigidity of the masseters was sufficient to prevent inspection of the throat. Usually the spasm is equal in the two masseters, but in one recorded case it commenced on the side that had been exposed to a draught (Harris and Doran). The spasm is always more extensive than the posture suggests; the muscles which are most powerful determine the attitude, but their weaker opponents also contract, perhaps equally. Thus the muscles that depress the jaw become rigid, as well as those that raise it. The jaw is sometimes fixed by both sets of muscles without being quite closed, and in one case the jaw remained widely open during a paroxysm.

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* From a plant Sardonia, so called because it grows in Sardinia.

Opisthotonic spasm is the rule, to which the exceptions are few. Rarely the trunk is bent forwards, from predominant cramp in the abdominal muscles and other flexors of the spine—"emprosthotonos." Still more rarely there is slight lateral flexion, "pleurothotonos," or the trunk and neck are rigid in a straight line, "orthotonos." These irregular forms seem to be rather more frequent in cases of idiopathic tetanus than in the traumatic form. In a case recorded by Treves considerable pain in the abdomen accompanied the emprosthotonos, and shifted to one side as the spasm became pleurothotonic.

The tonic spasm, although constant, usually varies in degree, and is occasionally, usually in slight cases, intermittent. Rarely there are no paroxysmal exacerbations. The paroxysms are sometimes brief and frequent, so as to resemble a slow clonic spasm. The tonic has been known to commence by clonic spasm.

When the muscles of respiration share the tonic spasm, the respiratory movements are limited in range, and the breathing is short and hurried. During severe paroxysms it is arrested, and death from asphyxia sometimes results. It is said that the spasm is inspiratory in some cases, in others expiratory. The former is probably the more frequent.*

During sleep, whether spontaneous or induced by chloroform, the spasm usually ceases, but it returns in undiminished force when the patient awakes. It has been thought, indeed, that after prolonged sleep from chloroform the violence of the spasms is increased in proportion to the length of time they have been kept in abeyance. Consciousness is unaffected, even to the last.

The pulse is increased in frequency, especially during the paroxysms, and is often very small. There is some reason to believe that the small size of the pulse is due to general vaso-motor spasm. The temperature varies much in different cases. It is often normal, or nearly so, during the whole course of the disease, although in fatal cases there is usually a moderate rise (2° or 3°) towards the end. In other cases there is a continuous elevation of 3° to 5°, without evening remission, although frequent measurements may show a slight rise at each paroxysm, and a slight fall at each interval, so that the chart presents a serrated tracing (H. C. Wood). In other cases irregular variations occur, without corresponding variations in the symptoms. Lastly, in some instances the temperature rises towards the end to an extreme degree, 108° or 110°, and, as Wunderlich first showed, the elevation may continue for a few hours after death, and even reach 114°. Some of the increase in body-heat may be due to the increased muscular work, but this cannot be regarded as the chief source of the pyrexia. Verneuil has pointed out that the cases in which the muscular spasm is greatest are rarely those in which the temperature is highest. Most of the fever is apparently of nervous origin. The hyperpyrexia is comparable to that met with in some cases of disease of the pons Varolii and upper part of the spinal cord, and may be associated with the fact that the earliest symptoms of tetanus proceed from this region.

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* According to Richet ('Société de Biologie,' March 4th, 1876), in the inspiratory spasm the glottis is open, in the expiratory spasm it is closed, and the latter entails more interference with circulation, and more danger of death from asphyxia, than the former. But in the case recorded by Harris and Doran ('Path. Trans.,' vol xxxi) there were severe paroxysms of inspiratory spasm, and the patient died asphyxiated at the end of the second day.

The urine is usually scanty and high-coloured, perhaps partly in consequence of the loss of water by the skin. The amount of nitrogenous matter excreted (urea, kreatinin, &c.) is not increased, even in pyrexial cases—a striking difference from most febrile diseases (Senator). Micturition may be interfered with by the spasm; sometimes there may be actual retention. The bowels also are usually confined, in some cases in very obstinate degree.

VARIETIES.—In their developed symptoms, cases of tetanus present little variety, but the early aspect of the case differs according to the seat of the spasm. There is, however, one form that presents considerable variation from the ordinary type, on account of the distribution and character of the symptoms, and also on account of its cause; it has been termed by Rose "cephalic tetanus" (Kopftetanus). It results from wounds on the head, chiefly in the region of the fifth nerve, and especially on the face. In many cases the wound has been due to a fall on the ground, or has been inflicted by an object recently in contact with the earth.* The chief peculiarity is that initial trismus is associated with paralysis of the face on the same side as the injury, and often with some tonic spasm on the other side. In many cases there is also a peculiar pharyngeal spasm on swallowing, often accompanied by respiratory spasm, or by a violent general tetanic paroxysm. This symptom resembles the spasm that occurs in hydrophobia, and hence the variety has also been termed "tetanus hydrophobicus." In severe paroxysms there may be turgidity of the veins of the head and neck, prominence of the eyeballs, and loss of consciousness. Such attacks, at first produced only by the attempt to swallow, may afterwards become spontaneous. The facial palsy usually involves all parts, as in disease of the facial nerve, but its mechanism is unknown. It may be preceded by a slight sense of "numbness" or tingling in some parts of the face, sometimes on both sides. There is no degenerative reaction during life,† and no disease of the nerve has been found after death; hence the paralysis is presumed to be of reflex origin. We have seen (p. 200) that irritation of the fifth nerve may cause paralytic ptosis, and it is worthy of note that in one recorded case ptosis formed part of the symptoms of this disease;[*] while in the remarkable case recorded by Roberts and Williamson there was complete palsy of all the muscles of the eye on the injured side, with motionless pupil, and on the other side, paralysis of the upper part of the face, and of the third and fourth nerves, with a sort of cataleptoid state of the upper lid. A few recorded cases have recovered, between the ages of $2\frac{1}{2}$ and 25, but those over 25 (which have occurred at various ages up to 52) have been fatal. In the cases that have recovered, the tetanic spasm and facial palsy have very slowly passed away, the symptoms continuing altogether several weeks. Death has usually occurred in the course of the second or third week.

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* A few instances have been recorded in this country, as by C. J. Bond ('Brit. Med. Journ.,' Nov. 10th, 1883), Nankivell ('Lancet,' July 14th, 1883), and Roberts and Williamson ('Lancet,' 1891).

† Bernhardt, Remak. Increased irritability of nerve and muscle, to both currents and to mechanical stimulation, was found by Nerlich, but a lowered irritability in the nerve by Von Spenje.

Other varieties depend on differences in the course of the disease and in the distribution of the early symptoms. In the most severe cases the spasm in the trunk may come on with that in the jaw; paroxysms occur almost from the first, and the patient may die in two or three days, sometimes in a few hours, and a still shorter duration has been known (see p. 677). On the other hand, the rigidity in the jaw and neck may exist alone for several days, even for a week, and the tetanic spasms on the trunk slowly supervene. In extremely rare cases transient spasm of the neck and jaw may be the only manifestation of the disease—the *abortive form* of Kussmaul.

In cases that recover, the disease always ends gradually, passing into a chronic stage. The paroxysms of spasm, at the end of one, two, or three weeks, become slighter and less frequent, and ultimately cease, while the tonic rigidity continues, slowly lessening in degree. It disappears first in the parts affected last, and endures longest where it commenced, in the neck and jaw, becoming occasional, and finally ceasing. Very rarely the tonic spasm passes off while the paroxysms continue. There is no special tendency to recurrence, for second attacks are at present unknown.[†] Nor have sequelæ (paralysis, &c.) been hitherto observed.

The *duration*, in fatal cases is, as a rule, less than a fortnight, and as already stated, frequently only a few days. After two weeks the patient's chance of recovery is fairly good.

The *mortality* of tetanus is extremely high, rivalled by few acute diseases, and excelled among its congeners only by hydrophobia. In traumatic tetanus the mortality is nearly 90 per cent., and seems to be rather greater in women than in men.[‡] The influence of age on mortality is uncertain. Adequate statistics for its determination do not at present exist. It is greater after severe injuries than after those that were slight. When the symptoms do not commence until after ten days from the receipt of the injury, the proportion of recoveries is much greater than in those that commence during the first ten days, and seems to increase, the longer the interval. When the disease begins within ten days, recoveries are extremely rare, and the mortality seems not to be influenced by the date of onset. Still, cases have been known to recover in which the disease commenced within three days from the time of the injury. The variations are doubtless related chiefly to the quantity of the toxic agent that has been inoculated, and perhaps in part to its state. Traumatic tetanus, although more common, does not appear to be more fatal in hot climates. At Calcutta it is said to be about 83 per cent. (Wallace).

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[*] Sereins, 'L'Un. méd.,' No. 173, 1886. The case is evidently an instance of this disease although it is not described as such.

[†] The case recorded by Ogle ('British and Foreign Med.-Chir. Rev.,' 1868, Oct., p. 488) is inconclusive.

[‡] It has been strangely understated by those who, disregarding the caution given

In the recorded cases of idiopathic tetanus which I have compared the mortality has been 50 per cent., and this is probably not far from the actual fact.[*] In hot climates, idiopathic tetanus is usually stated to be more fatal than the traumatic variety, but the statistics of Wallace give a mortality among males of 56 per cent.[†]

The mortality in tetanus neonatorum is extremely high, probably at least as high as in traumatic tetanus. Of all forms, however, puerperal tetanus is that which is most frequently fatal. When it occurs after abortion, recovery is practically unknown. The only recorded case which had not ended in death was still in progress at the time of the report.[‡] After labour, all the cases in which the disease could be referred only to the process of parturition have been fatal. Of three cases that recovered, in one eclampsia occurred before childbirth, and in the other two the tetanus was apparently excited by exposure to cold, in one four days, in the other three weeks after parturition.

Causes of Death.—In about two thirds of the fatal cases of tetanus death occurs during a paroxysm, either from failure of the heart, or from asphyxia in consequence of the prolonged arrest of respiration. The cause of cardiac failure is supposed to be the increase of the intra-vascular pressure (partly by the compression of vessels by the contracted muscles, partly by vaso-motor spasm) to such a point that the enfeebled heart is unable to contract. The severity of the strain

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long ago by Lawrie, have estimated the mortality from collections of cases which have been separately published, most of them *because* the patients recovered. For instance, the total mortality was estimated by Yandell ('Brain,' 1879) from a collection of 449 cases to be 46 per cent.! The 164 cases of traumatic tetanus contained in the consecutive series of Lawrie, and of Poland and Taylor, comprise 140 males, of whom 122 died (87 per cent.), and 24 females, of whom 22 died (91 per cent.).

[*] This conclusion agrees with that of the idiopathic cases in the series of Lawrie, Poland, and Taylor. Of these, 11 in number, 6 died.

[†] Wallace, "Statistics of Tetanus in the College Hospital, Calcutta" ('Indian Med. Gazette,' Jan. 1st, 1881). Of 93 males, 53 died. The statistics regarding females are not available, because puerperal cases are included in the idiopathic form.

[‡] A case described by Lawrie in a letter to Simpson, and published by the latter, loc. cit.

to which the heart is exposed is shown by one case in which a degenerated wall gave way during a violent paroxysm.* In the remaining cases death occurs from exhaustion, or from some accidental complication.†

PATHOLOGICAL ANATOMY.—Rigor mortis usually sets in early, and is well marked. It is, indeed, said that the spasm during life may pass into the rigidity of death, but in most cases brief relaxation occurs.

The lungs are usually congested, and there may be œdema, hypostatic pneumonia, local emphysema, and subpleural extravasations—direct or indirect results of the interference with the pulmonary circulation by the frequent and violent spasm. The heart is sometimes relaxed; more frequently it is firmly contracted, probably only from rigor mortis. The liver and spleen are usually anæmic. The kidneys may be pale or congested; sometimes they contain extravasations. The difference in the amount of congestion depends chiefly on the mode of death, whether from exhaustion or during a spasm which arrests the movement of the blood.

The muscles often contain small extravasations. Rupture of individual fibres may be found on microscopical examination, and occasionally a large muscle may be torn across by the violence of the spasm. Such rupture has only been seen in the flexors of the trunk and hip (rectus abdominis frequently, psoas rarely‡), which are contracted, and at the same time are stretched by the more powerful spasm in the extensors. The fibres, under the microscope, usually present a normal appearance, but occasionally granular degeneration is seen, or there is a tendency to split up longitudinally (Bowman). Chemical analysis has shown that they contain more water than normal, and less albuminous material, but in the alcoholic extract there is more nitrogenous matter and a substance containing phosphorus—lecithin (Danilewsky).

In traumatic cases the wound may be in almost any condition, healthy or unhealthy, in process of normal cicatrisation, or even actually healed. Perfect cicatrisation is necessarily rare, on account of the period at which tetanus usually commences. Otherwise no state of wound is sufficiently frequent to be of significance. The nerves of the part injured have presented, in the majority of cases, no abnormal condition that could be recognised even by the microscope. In some cases, however, distinct evidence of inflammation has been found, the nerve being swollen and reddened, and, in a few instances, ascending neuritis has been traced up the nerve, even as high as the cord—reddened nodular swellings, usually separated by normal interspaces.

* Duclaux, 'Bull. de Thérapeutique,' 1877, Sept. 30th.

† *E. g.* in a case recorded by Pitram (' Wien. med. Presse,' Nov. 1st, 1886) death was due to pulmonary embolism from a clot in the hypogastric vein, the formation of which was supposed to be due to the compression of the vein by the muscular spasm.

‡ Wynne Foote, Earle.

Increase of interstitial tissue, with degenerative changes in the nerve-fibres, is found in such cases. It is certain, however, that local neuritis is no constant part of the morbid appearances in tetanus. The facts relating to the micro-organisms will be described in the section on the Pathology of the disease.

In puerperal tetanus there is no constant abnormal appearance in the uterus, but in some cases metritis, and decomposing remnants of the placenta, have been discovered.

In newly-born children, as we have seen, the umbilicus may be inflamed, and an arteritis umbilicalis may be traced along the cord within the abdomen. The adjacent peritoneum is often also inflamed. In some cases, however, these parts are normal.

In the brain and spinal cord the only common changes in tetanus are distension of vessels, large and small, and the presence of minute hæmorrhages, such as are met with in all cases of death from convulsion, and are perhaps produced chiefly during the process of death. In many cases, no morbid appearance has been discovered on microscropical examination.* In some, slight changes have been found, but these have varied much. Many are of no significance, such as an unusual amount of interstitial tissue in the white columns, spaces around the vessels, yellow pigmentation of the ganglion-cells, amorphous exudation at the bottom of the fissures, and an increase of nuclei around the central canal, sometimes obliterating it or extending laterally in the posterior commissure.

The microscopical changes which have been observed in the cord,† and are possibly or probably connected with the disease (besides the vascular distension and minute extravasations), are—increase of the corpuscles in the interstitial tissue and around the vessels, chiefly in the grey substance; areas of "granular disintegration," sometimes amounting to actual cavities containing amorphous or finely granular material, also chiefly found in the grey matter; irregular areas of carmine-staining amorphous "exudation" in the grey matter and posterior columns, and various changes in the large ganglion-cells, swelling, with indistinctness of the outline of the cell and of the nucleus; shrinking of the cells; apparent destruction of the processes. Similar alterations have been found in a few cases in the medulla oblongata, but slighter in degree. These changes, when found, have presented no uniformity in character or distribution, nor can any relation be traced between their position and the seat of the wound.

PATHOLOGY.—The chief fact of the pathology of tetanus is its relation to the specific organisms, which has been mentioned in the section on Etiology. The discovery is an event of the last six years, and, as regards the traumatic and infantile forms, is established by overwhelming evidence. It places tetanus distinctly in the rank of "acute infective diseases;" infective, however, chiefly, perhaps exclusively, by inoculation. The discovery has been facilitated by the special susceptibility to tetanus possessed by certain animals, especially mice, rats, guinea-pigs, and rabbits. As long ago as 1884 it was found (by Carle and Rattone) that the disease could be produced in rabbits by inoculation with the pus of the wound of a person suffering from tetanus;[*] and, independently, Nicolaier found that the disease was produced in the animals mentioned above by inoculation with different kinds of earth, and that a peculiar bacillus was always to be found in the active earth, and also in the wound in which it had been inoculated. In 1885 Rosenbach found that the same bacillus was always present in the secretion and tissues of the wound which had given rise to traumatic tetanus in man; when the wound was due to a fall on the earth, he found the same organisms in the soil at the place, and he confirmed the observation that inoculation with the pus and tissues of the wound in man gave rise to the disease in animals. He cultivated the bacillus, and proved that it was then equally effective. These facts have been since confirmed by many investigators, and may be accepted as invariably true. The disease has also been produced in horses, sheep, and sometimes in dogs, but the last are not prone to tetanus, and require a large dose.

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* As in four cases examined by F. Schultze ('Neurologisches Centralblatt,' 1882, No. 6), two by Hadden ('Brain,' Oct., 1885), and five by Bowlby ('St. Bart. Hosp. Rep.,' 1884).

† Chiefly by Lockhart Clarke, Clifford Allbutt, Ross, Doran, Harris, Dickinson, and Aufrecht.

The bacillus is met with in two forms,—fine needle-shaped rods, and similar rods with a much wider oval "head" at one extremity, containing spores. The former are 4 to 6 μ[†] in length, the latter 6 to 8. The "head" is 1·5 μ in width, the rod only half as wide. They are always found in the tissues of the wound, and have sometimes been met with sparingly in the blood and spinal cord, but not in other organs. They possess remarkable power of resisting heat, even exposure to 175° F. for an hour,[‡] and their spores for six hours;[§] by this they may be separated from other organisms which are killed. But they cease to form spores at 108° F. They are "anaërobic," growing in an atmosphere of hydrogen or *in vacuo*, and are slowly weakened and at last killed by free access of air, and especially of light. They are destroyed by nascent chlorine, and by a 5 per cent. solution of carbolic acid, but not by weaker solutions. The chief habitat of the organism is the soil, in which they have been found so widely distributed as to be found in earth taken, for instance, from various parts of Warsaw, and in sixteen out of twenty-three specimens from various parts of Copenhagen. The vital endurance of the organisms, and especially of their spores, under favorable conditions is great. The dried pus from the wound of a tetanic horse has been found active after sixteen months,[*] and earth in which tetanus spores had been placed was virulent at the end of three years.[†]

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[*] Buchanan, 'Glasgow Med. Journ.,' 1890.

[†] Or $\frac{1}{6000}$ to $\frac{1}{4000}$ in.

[‡] Kitasato, 'Zeitsch. f. Hyg.,' Bd. vii.

[§] Schwarz, R., 'Arch. Sc. Med.,' 1891.

On account of the small number of bacilli found within the body it was suggested, first by Nicolaier, that the symptoms are due, not to the organisms themselves, but to a strychnia-like toxine produced by them; and this opinion has been generally accepted, and it has been shown to be correct. Vaillard and Vincent[‡] succeeded, by a special process of filtration, in separating the bacilli from the material they had formed, and found that the former were inert, while the latter at once caused the disease. Brieger,[§] indeed, had previously described four distinct forms of toxic material as produced by the bacilli, of which three cause symptoms of tetanus, and for the most active of these he proposed the name of "tetanin." It has been thought that the symptoms of traumatic tetanus are due to the toxine which is inoculated with the bacilli; this is probably true when the affection begins in a few hours, but the interval which usually elapses suggests that the virus is formed by the organisms either in the wound or in the blood.

These discoveries have naturally suggested attempts to produce immunity by methods analogous to those employed by Pasteur in the case of hydrophobia, but hitherto with only limited success. Tizzoni succeeded in the case of dogs, who are little prone to tetanus, by a series of inoculations with the cultivated virus, at first minute, and gradually increasing in strength; and he found that their blood-serum destroyed the activity of the virus—in consequence, he believes, of the presence of a peculiar albuminoid body which he terms "tetanus antitoxine:" its power is destroyed by lactic and hydrochloric acids, and by a temperature of 150° F. He failed to produce immunity in rabbits and guinea-pigs, or to arrest the developed disease except in rats. Arrest was also obtained by Behring and Kitasato, who found that simple blood-serum, without the corpuscles, produced immunity, and that the serum of the animals rendered immune was even more potent.

We have seen that the intensity of the cause of tetanus can be varied experimentally, and probably similar variations exist in that which enters accidental wounds. That which is found in soil manured and decomposing, is said to be especially intense. The presence of the organisms of decomposition has been found to favour the growth of the tetanus bacilli, which ultimately persist while the others perish. These variations, and differences in the quantity of the virus that enters the wound, probably cause the variations in the intensity of the disease, and the corresponding differences in the period of incubation. What part is played by individual predisposition is unknown (see Etiology).

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[*] Kitt, 'Cent. f. Bact.,' 1889.

[†] Raume, 'Zeitschr. f. Hygiene,' Bd. vii.

[‡] 'Comptes Rend.,' 1891.

[§] 'Deut. med. Wochenschr.,' 1887.

To what part of the central nervous system is the disturbance to be referred which is produced by the poison? The early symptoms are in the region supplied by nerves that arise from the highest part of the spinal cord, the medulla oblongata, and the pons, and must be referred to centres in this region. The same indication is afforded by the co-ordinated respiratory spasm that forms so conspicuous a feature in the attacks, by the vaso-motor spasm, by the occasional hyperpyrexia, by the phenomena of cephalic tetanus, and by such cases as one recorded by Silbermann, in which a fall on the back of the head was followed in a few hours by symptoms of tetanus, accompanied by striking cyanosis and dyspnœa. Permanent trismus sometimes results from organic disease of the pons Varolii, and general tonic spasm, especially marked in the legs, is not uncommon from hæmorrhage in this situation. It is only in the later stage of tetanus that there is evidence (in increased reflex excitability) of a morbid state of the lower spinal centres, which, secondary in point of time, is doubtless secondary in its production. The symptoms point, therefore, to the pons and medulla as the seat of the chief disturbance of nerve-function in the disease.

The facts that the injury causing tetanus often involves the nerves, and that these are sometimes inflamed, and even the seat of an ascending neuritis, must not be overlooked in the light of the new pathology. The tetanic spasms sometimes start from the seat of the injury, and in cephalic tetanus the injury in the region of the fifth nerve causes paralysis of motor nerves related to it in reflex action, although the specific virus is present in the wound in this as in other forms. These facts suggest that the toxine, which acts so powerfully on the central nervous system, has also a local action on peripheral nerves to which it can gain access.

In connection with the theory of nerve irritation a case recorded by Terrier* deserves especial note. An injured toe had become gangrenous, and was amputated. Trismus, absent before, was present a quarter of an hour afterwards, and the patient averred that the pain of the operation distinctly made his jaw muscles contract while the amputation was being performed. He died from the tetanus in two days. Here it would seem that the toxic irritation brought the centre into a condition so unstable that its discharge was produced by the additional stimulus. Verneuil has also noted that surgical interference with a wound may apparently excite tetanus. It may be that a similar influence is exerted by exposure to cold. In the unstable condition of the centres, a slight degree of surface chill may cause discharge of the nerve-cells. For instance, in the case of tetanus after ovariotomy, recorded by Harris and Doran, the contraction commenced in the masseter on the side turned towards a window, the weather being very cold at the time. That the surface chill causes a peculiar nervous stimulation is the more probable, since we know that it causes other effects through the agency of the nervous system, stimulating, for instance, the vaso-motor centre to constrict the vessels of the skin, and even the motor centres to produce the muscular spasm of "shivering," in which the muscles of mastication always take a conspicuous part. It is noteworthy, however, that, whether acting alone or conjointly, the effect of cold is always rapidly produced. Rarely more than twenty-four hours pass before the appearance of the first symptoms. Lastly, it may be observed that in tetany we have a disease consisting of tonic spasm, and sometimes caused by cold.

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* Terrier, 'Gaz. des Hôp.,' 1874.

In true tetanus neonatorum, the pathology of the disease is the same as in the traumatic form, the bacilli having been invariably found in the umbilical wound. The precise nature of puerperal tetanus and of the idiopathic form has still to be ascertained. If, as previously suggested, they are produced by the specific organisms, it is probably by the inhalation of spores from the air. The organisms seem to have no effect when taken into the stomach.

Diagnosis.—The symptoms of tetanus are so peculiar and so pronounced, that the diagnosis rarely presents any difficulty, except in the earliest stage of cases that commence insidiously. Under such circumstances the stiffness of the neck may be mistaken for muscular rheumatism, the more readily if the symptom has followed exposure to cold. But the existence of rigidity in the muscles of the jaw, never present in simple rheumatism and rarely absent in tetanus, should at once arouse suspicion. In the rare cases in which difficulty in swallowing precedes trismus, the nature of the case can only be suspected from the facts that there is no local affection to account for the symptom, no evidence elsewhere of paralysis, and no history of any cause of hydrophobia, while trismus and tetanic spasms usually follow within twenty-four hours.

The characters of the developed disease resemble those of strychnine poisoning more than any other condition. A mistake is rare, but has probably been made in one or two recorded instances. In strychnine poisoning the symptoms never commence by trismus; they come on and develop in a more rapid manner than has ever been seen in tetanus except in traumatic cases, in which the wound assists the diagnosis. The reflex excitability is an early symptom in strychnia poisoning, but is late in tetanus, and the severe epigastric pain of the latter is absent in the former, in which there are often collateral circumstances to suggest poisoning.

In hydrophobia there is no initial rigidity in the jaw or elsewhere. The first paroxysms are of respiratory spasm, excited by attempts to swallow. These may be present, however, in the "hydrophobic" form of tetanus, and in it swallowing is difficult, but a wound on the head and facial palsy are conspicuous. Cases of hydrophobia in which there are tetanoid spasms in the later stage of the disease have been confused with tetanus, but attention to the initial symptoms will prevent error. These spasms are merely an excessive development of those that are frequently met with in hydrophobia, and do not indicate that the two diseases co-exist, as has sometimes been erroneously imagined.

In hysteria, tetanoid spasm is extremely rare except as part of a convulsive attack, and then its nature is sufficiently conspicuous. But trismus, causing persistent closure of the jaws, occurs in hysteria. It may succeed a convulsion, and last until another, or it may come on without obvious cause, continue for a few hours or days, and then suddenly vanish. It is prone to recur, and this character, the suddenness of onset, its complete degree, the absence of rigidity in the neck, and the presence of other symptoms of hysteria will rarely leave any doubt as to its nature. It should be remarked that the symptoms of tetanus, coming on after an injury or exposure to cold, must not be treated lightly because they occur in an hysterical person, or follow fear of the disease.

In tetany there is widely spread tonic spasm with paroxysmal exacerbations, but its distribution is characteristically different. The limbs are most affected towards the extremities, the arms more than the legs, the hands most of all, and trismus is a late, and not an early symptom. In each of these respects, the condition in tetanus is the reverse. Even in the most acute and violent cases of tetany a mistake can hardly be made, especially if attention is paid to the peculiar posture of the hands.

In many cases of tetanus there is considerable difficulty in determining whether it is to be regarded as traumatic or idiopathic. They are the cases in which tetanus is apparently excited by cold in a person who has had a trifling injury, perhaps some time before. The question is fortunately not of any great practical importance, and each case must be judged on its own merits. For scientific purposes we must include such cases in the traumatic variety.*

PROGNOSIS.—The statistics of the mortality in tetanus show how grave the prognosis is in every case, and it is still grave, however slight the initial symptoms may be, or however trifling the injury which has produced it. But it is distinctly worse if the injury is severe than if slight. After a lacerated wound, the compound fracture of a limb, or distinct contamination of the wound with earth, and also after labour, recovery is extremely rare. The chance of recovery is less if the first symptoms occur before the tenth day from the receipt of the injury. Most recoveries occur in cases which begin after the first ten days, and the prognosis is better the longer the interval. It is worse when the spasm quickly extends to the trunk, better if trismus exists alone for several days. After the first four or five days the prognosis improves with the duration of the disease, although it does not become decidedly hopeful unless the symptoms have lasted for a fortnight, or present a distinct and persistent diminution. It is doubtful whether the prospect of recovery is materially influenced by sex or age.* Adequate statistics to determine this point are not at present forthcoming. Previous intemperance lessens the chance of recovery, and so also does inability to swallow and considerable elevation of temperature, but its subsequent fall is not of significance if the symptoms continue. The concurrence of cold with injury as a cause does not influence the prognosis. Under the most favorable circumstances—in cases, for instance, which occur a fortnight after the receipt of a trifling injury—the probability of recovery is not more than equal to that of death. On the other hand, hardly any case of traumatic tetanus is absolutely hopeless. Cases occasionally recover under all conditions of cause and character.

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* By this means we run least risk of error. It is certain, however, that some proportion of cases of idiopathic tetanus will have a recent scratch or cut or bruise, without this being concerned in the production of the disease.

The prognosis in idiopathic tetanus is, in temperate climates, a little better than in the traumatic form. In hot climates it is regarded as even more grave. In tetanus neonatorum the prognosis is nearly the same as in the traumatic form in adults (provided the children are not exposed to powerful insanitary conditions), and here also the prognosis is better the longer the interval after birth before the symptoms appear. After abortion and labour, the chance of recovery is only appreciable when the disease is distinctly excited by cold.

TREATMENT.—For tetanus, as for most acute diseases, no specific remedy is known, although the recent discoveries give distinct promise that it may not be long before some method of counteracting the action of the bacilli is discovered. It will probably be possible to foresee the malady by microscopical examination of the pus in any wound contaminated with earth, and then prophylaxis, as in hydrophobia, may become possible. But for this we have yet to wait.

Tetanus is a disease of limited, though variable duration, and if the patient can be kept alive until it is over, he recovers. Moreover, there is reason to believe that the intensity of the disease can often be lessened by treatment, but the variable severity of the affection renders it very difficult to assess the influence of the remedies employed. Three elements in treatment have hitherto chiefly received attention—general management, operative treatment in traumatic cases, and the endeavour to relieve the symptoms by the use of drugs.

In general management, rest and food are the two essential measures. All possible sources of peripheral irritation should be avoided. The patient should be kept absolutely still, and the room darkened. Liquid nourishment should alone be given, even if the trismus is not complete. In most cases, by patience, a fair quantity can be taken through the closed teeth; sometimes there is a space between the teeth, and a tube can be introduced through which the food may be sucked. When this is impossible, sufficient relaxation of the spasm to permit of food being given may be obtained by the inhalation of chloroform, or liquids may be injected into the œsophagus by a long catheter passed through the nose. If this brings on spasm, it is better to employ peptonised injections into the rectum. A tooth has been extracted to permit feeding through a tube, but it is better, if possible, to avoid any fresh irritation of peripheral nerves. In infants the catheter passed through the nose has usually been employed.

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* Regarding sex, compare the mortality given on p. 684.

The surgical measures which have been adopted in traumatic cases comprehend amputation, excision of the cicatrix or wound, neurotomy, and nerve-stretching. Of all these it may be said that in most cases in which they have been employed they have apparently exerted no beneficial influence on the course of the disease. Indian experience of these measures is at least as unfavorable as that in England.* A curious case has indeed been described by Reichert† in which stretching of both sciatic nerves was followed by improvement and ultimate recovery; the cause of the tetanus was a bite on the back by a horse, but the patient was an epileptic. The *modus operandi* of the operation is difficult to understand. The common failure of these proceedings is rendered intelligible by what we now know of the pathology of the disease. A more promising measure would be the early excision of the wound in any case in which the bacilli can be found in it or in the soil of the place at which it was inflicted. In such a case, amputation may even prove justifiable. The late excision of the cicatrix has apparently been effective,‡ although it usually fails, no doubt because the toxine has invaded the whole system. Perhaps, also, division of the nerve is justifiable when tetanic spasms start from the injured part. Most of the cases in which local surgical treatment has had a distinct and immediate effect have been of this character. In one case, for instance, a small nerve in a wound was tender, and pressure upon it brought on severe tetanic spasms. The nerve was excised, and the spasms ceased. In another case, tetanus came on after a wound was healed; the cicatrix was tender, and pressure on it excited the spasm, which ceased after the cicatrix had been excised.

There are few diseases for which so many and such varied drugs have been employed as in the treatment of tetanus, but hitherto not one has been found to exert a powerful influence on the disease, not one that does not usually fail when the disease is severe, and frequently when it is moderate in intensity, and few that have not been credited with cures, chiefly in slight cases. Recovery has, indeed, in most cases been obviously due much more to the character of the attack than to the treatment employed. Still, there is reason to believe that drugs have, in many instances, helped recovery, and in some cases have actually turned the scale and saved the patient's life.

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* Wallace, loc. cit.

† 'Bayer. Aerztl. Intell.-bl.,' 1885, No. 5.

‡ Cotteril, 'Lancet,' 1888.

Of the agents employed there are some that give temporary relief to the spasm, and are used to afford rest or avert death, but which do not influence the course of the disease. The most efficient is the inhalation of chloroform. With complete narcosis, the spasm passes off, but it returns when the influence of the chloroform is over. Ether has a similar action, but is less convenient. Nitrite of amyl has been said to relieve spasm more speedily than chloroform, and H. C. Wood has recommended it as a most valuable agent for averting death during a paroxysm; at Guy's Hospital, when employed in quantities of ♏iij to ♏v, it was found that the spasm became more intense at first, although slighter afterwards. The continuous inhalation of chloroform does not seem to modify the course of the disease, nor has any benefit resulted from attempts to charge the air of the room with the vapour of chloroform.*

Sedative drugs have been employed in the treatment of tetanus in two ways, occasionally, to procure sleep, and continuously, to lessen the severity of the spasm. In severe cases, it is rare that a marked effect is produced by any drug, but in cases of moderate severity, distinct amelioration has been obtained by many agents. Bromide of potassium has been given at frequent intervals in large doses, four to six drachms daily, and the spasm has been observed to increase when it is discontinued, and to lessen when it is resumed (Southey). It may, perhaps, with advantage be injected into the rectum with food; Nothnagel has found that it tends to cause ascending contractions in the bowel, and thus an injection is carried higher up, and is rapidly and more completely absorbed.

Chloral hydrate has been largely substituted for the administration of chloroform by the mouth, although it does not remove severe spasm so completely as the inhalation of chloroform. It may be given at night to procure sleep, and frequently succeeds, or it may be given continuously, and has thus, in many cases, appeared distinctly beneficial. Indeed, it has been held to have more influence on the disease than any other drug, by a very large number of observers.† Large doses must be employed. Verneuil, for instance, who is a strong advocate of its use, would never give, to an adult, a smaller dose than ʒj, or less, in the day, than ʒiv, and he has sometimes given ʒiij at a dose and ʒvij in the day. It needs to be continued for a considerable time. In two cases which recovered, ℥viij were given in the course of a month. Antipyrine was used in conjunction with chloral in one idiopathic case which recovered. Chloral has been injected into the veins by Oré, but this is a dangerous proceeding, since it has caused extensive thrombosis in several cases (Lannelongue and others). In tetanus neonatorum it has usually been given in doses of one grain, but this is probably too small. Opium and morphia have been largely used, the latter by subcutaneous injection; it is useful for the purpose of procuring sleep, in doses of a quarter or half a grain, which may be employed in addition to the continuous administration of bromide and chloral. The continuous use of morphia has not, on the whole, been so often effective as that of other sedatives, but it has seemed to cure some cases, one of idiopathic tetanus,[*] and others of the traumatic form.[†]

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* Simonin used in this way 22 kilogrammes of chloroform without effect.

† Schmidt ('Bayer. Intell.-bl.,' 1885, p. 329) records the recovery of four out of five cases treated with chloral, but so favorable an experience must be regarded as altogether exceptional, and perhaps open to suspicion.

Belladonna, atropine, Indian hemp, and aconite have also been frequently employed. Under their use (as under that of all other drugs) recoveries have been occasionally observed, but on the whole their utility has been even less frequently recognisable that that of other sedatives. The same may be said of conium, lobelia, nicotine, and tobacco, veratrum viride and gelsemium.[‡]

Next to bromide and chloral, Calabar bean has received most praise. The extractum physostigmatis has been usually employed subcutaneously, in doses of from one third of a grain to one or two grains, or by the mouth, one to four grains. Still larger doses have been sometimes given by the mouth, *e. g.* seventy-two grains in twenty-four hours, and 1026 grains in forty-three days, in a case that recovered.[§] Toxic effects are not easily produced, and the pupil does not contract as it does in health, even when nausea and feebleness of pulse have been produced. Eilert has suggested that local effects may be lessened by the simultaneous injection of atropine. In infants, from one thirtieth to one third of a grain has been given under the skin. The salicylate of eserine had a marked influence on the spasms, injected in doses of $\frac{1}{100}$ grain, in a case in which chloral and bromide had previously been used. The tetanus began on the fifth day after the removal of a tumour of the leg, and the patient recovered.[‖] Jaborandi and pilocarpine have been used, generally without success, although three recoveries have been described by Casati.[¶]

The alleged success of curara in hydrophobia has led to its use in tetanus, but seldom with effect. Small doses, $\frac{1}{100}$ grain, do not influence the disease. With large doses up to half a grain every hour, the muscular spasms become slighter, but life has rarely been saved. In one case it was pushed until the patient passed into a state of collapse and respiration ceased. Prolonged artificial respiration, with faradisation of the phrenic nerve, revived the patient; the spasms remained absent for several hours, and then returned in slighter form, and the patient recovered. Spasm is probably lessened by the influence of curara on the intra-muscular nerve-endings, as it is not known to have any influence on the spinal cord. It appears only to avert death from spasm by bringing the patient equally near to death from paralysis.[*] Among other drugs which have been used are iodide of potassium, carbonate of iron, arsenic, antimony, mercury, urethane, and strychnine. Cases have recovered in which each has been used, even, strange to say, the last, which distinctly increases the spasm.[†]

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[*] F. J. Smith, 'Lancet,' 1889.

[†] C. Dukes, 'Lancet,' 1888; Morrison, ib.

[‡] Four cases are said to have been successfully treated by veratrum viride and gelsemium by R. B. Harris ('New York Med. Record,' 1884, July 12th).

[§] Watson, 'Practitioner,' April, 1870. For another successful case see Dougall, 'Glasgow Med. Journ.,' March, 1885.

[‖] Rees and Raywood, 'Lancet,' 1889, ii.

[¶] See 'Brit. Med. Journ.,' 1889, quoted from 'Raccoglitore Medico.'

External applications, chloroform, aconite, &c., to the affected part appear useless, but applied to the epigastrium they sometimes give relief to the severe pain often felt there. Cold to the spine (ice, ether spray) has been employed without distinct influence on the spasm. Electricity (the voltaic current from the spine to the muscles) has been observed to lessen the spasm in trifling cases, but probably did not influence the course of the disease. Continuous warm baths have been occasionally employed, since the time of Ambrose Paré. Zechmeister kept one patient in a bath for a fortnight, but the treatment, used in eight cases, did not influence the mortality, since seven of the patients died. It seemed, however, to moderate a little the intensity of the spasm. Bleeding was formerly largely used, and, opposed as the measure is to the therapeutic principles of the present day, it may remove some of the toxine from the system; at any rate, cases have recovered, and the flow of blood has appeared, in some instances, to lessen the spasm. Transfusion of blood has been once employed, but without success (Sakler). No case has yet been influenced by the injection of the serum of immune rabbits, supposed to contain the antitoxine. It was tried in a case of tetanus neonatorum, but four injections had no effect.[‡]

On the whole, the drugs that seem to deserve most confidence in the treatment of tetanus are bromide of potassium and chloral, morphia, and perhaps eserine. The first two may with advantage be combined, the influence of both being kept up continuously, or bromide may be given frequently, and chloral in occasional hypnotic doses. If the violence of the spasm threatens death during a paroxysm, this should be, if possible, averted by the inhalation of chloroform or nitrite of amyl, but on the value of the latter for this purpose further observations are necessary. Lastly, it is of great importance, in the event of apparent death during a paroxysm, that an attempt should be made to restore the patient by artificial respiration. It is surprising that this measure has been so rarely adopted. Its importance is illustrated by a case recorded by Farrage.* During a severe paroxysm, heart and respiration stopped, the patient was apparently dead. Artificial respiration was employed, and in five minutes the heart's pulsations could be again perceived. For two days the spasm was extremely slight; a most violent paroxysm then occurred, during which the patient died, no medical assistance being at hand. The remarkable freedom from spasm in this case, and one or two others on record, after revival by artificial respiration from apparent death, can only be ascribed to the influence of the arrest of the circulation on the morbid condition of the centre. This fact, and the evidence that the primary disturbance is in the medulla oblongata, suggest the desirability of trying the effect of ligature of the vertebral arteries, which Alexander has introduced for the treatment of epilepsy. The operation is severe when both vertebrals are simultaneously tied, but in a case in which the danger was extreme it might be justifiable.

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[*] Karg ('Arch. f. klin. Chirurg.,' xxix, p. 338) relates four cases in which the transient amelioration of the symptoms did not prevent death. He advises the simultaneous use of morphia. One noteworthy lesson from his cases is that if artificial respiration becomes necessary it is *not* facilitated by tracheotomy. Cases successfully treated have been recorded by Berckham ('Berl. kl. Wochenschr.,' 1884, No. 48) and Gontermann (ib., 1883, No. 44). In the latter case, the tetanus followed an injury to the head. Nine injections were given in the course of a few days, each consisting of ¼—⅙ grain of curara, dissolved in 40 parts of water and 2 of spirit. He makes the reasonable suggestion that the activity of the specimen employed should always be proved by an injection in an animal.

[†] A collection of 937 reported cases, beginning before the fifteenth day, has been made by Melden, to compare the apparent effect of different drugs, but it is doubtful what value can be attached to the statistics of so very large a number of cases. Chloral and curara are each credited with the cure of a quarter of the cases in which they were used; opium with one twenty-fifth; conium one seventh; Indian hemp a sixth; bromide a fourteenth; alcohol a quarter; and a third of those treated by other agents are said to have recovered ('Lancet,' 1887).

[‡] Babinsky, 'Berl. kl. Wochensch.,' 1891.

TETANY.

The condition designated "tetany" is characterised by tonic muscular spasm or "contracture," affecting especially the extremities, symmetrical in distribution, and either paroxysmal or continuous. The spasm is often preceded and accompanied by sensory symptoms, tingling, formication, or pain.

The affection was first described more than fifty years ago by Steinheim in Germany, and Dance in France, but, like so many other maladies, it was through the study and description of the disease by Trousseau that it became generally known. Trousseau called it "tetanilla," but the name "tetany," by which it is now universally known, was suggested by Lucien Corvisart in 1852.†

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* 'Lancet,' 1860, Sept. 18th.

† Trousseau's account of the disease is accessible to English readers in the volume of lectures translated by Bazire. Other descriptions have been given by Riegel ('Deut. Arch. f. kl. Med.,' xii, 1863, 405); Weiss ('Volkmann's klin. Vort.,' No. 169); Buzzard ('Clin. Lect.,' p. 411); and Abercrombie ('On Tetany in Young Children,' London, 1880).

Causes.—Tetany is rather more frequent in males than in females, the proportion being as seven to six, but this relation does not obtain at all ages. It occurs at all periods of life, from infancy to old age, but is most frequent in early childhood and in early adult life. Of 150 cases that I have collected from various sources or observed myself, 142 are available for comparison on these points.

Ages . .	1–4	5–9	1–9	10–19	20–29	30–39	40–49	50–61	
Males .	26	5	31	23	9	4	5	4	= 76 Males.
Females .	8	3	11	13	15	19	8	0	= 66 Females.
Total. .	34	8	42	36	24	23	13	4	=142

Thus the disease is most frequent in infancy and in the second decade of life. More than half the cases occur during the first twenty years. In early childhood the disease is far more frequent in males than in females, but between the ages of twenty and fifty the liability of the sexes is reversed, and females suffer twice as frequently as males. Over fifty, on the other hand, the only recorded cases have been in males. These figures correspond, on the whole, with those supplied by smaller series of cases lately published, but some variations exist, owing to special features that have marked periods of special prevalence. Young children, for instance, have sometimes been the only sufferers, and in a number of such cases at Prague, the proportion of males to females was only as 2 to 1, while of a series of 41 cases in adults, 36 were males and 5 females.

A neuropathic heredity can be traced in only a small proportion of cases, but that a family predisposition sometimes exists is shown by instances recorded by Abercrombie, in which there occurred, at different times, in each of two families four cases, and in another family three cases. It has often been observed to be more frequent in winter or in spring than in summer or autumn, in the case both of outbreaks and of recurrences.

Most of the male cases have occurred in the lower classes, and occupation is influential partly as involving exposure to cold and fatigue; but the affection, especially in Germany, is common in shoemakers and tailors, perhaps from some influence exerted by posture combined with that of a sedentary life.

A direct exciting cause of tetany can be traced in at least three quarters of the cases. The most frequent is diarrhœa, usually long continued and exhausting, but sometimes acute and brief. Epidemic diarrhœa has been the cause of some local outbreaks of tetany in young children. It often co-exists with other causes. Next in frequency is exposure to cold, which may produce also catarrhal disturbance. A lady a month after confinement caught cold in driving; an attack of tonsillitis came on, and, in a few days, a sharp attack of tetany, quickly yielding, however, to treatment. Cold is effective, especially when conjoined with fatigue, acute disease, and lactation. A series of cases in women who were suckling led Trousseau at first to propose for the disease the name of "nurse's contracture." The comparative frequency of the disease in adult women is almost entirely due to the various influences of maternity. The tetany may also come on during pregnancy, usually during the second half, but sometimes early; it almost always continues until delivery and then ceases, at once, or in a week or two. In those who have suffered before marriage it is especially apt to occur during pregnancy, often more than once, but not always in successive pregnancies. It may also develop, in rare instances, after confinement. Other occasional causes are simple anæmia, prolonged muscular effort of various kinds, alcoholism, and sexual excess, especially masturbation. Many cases have been observed in the subjects of dilatation of the stomach,* sometimes with ulceration or with the cicatrix of an ulcer, which in some has been at the middle of the organ, associated with external adhesions, an hour-glass form of the organ, and even with rotation of the pyloric end.

In young children the indications of rickets are rarely absent, and in them tetany is evidently allied to the carpo-pedal contractions so common in that disease, and it is often associated with laryngismus stridulus and convulsions. In three recorded instances the disease was apparently due to intestinal worms (tæniæ), and ceased when these were expelled. It very rarely follows a fright, and seldom an injury, but there is one operation after which it is singularly frequent,—excision of the thyroid. When the whole thyroid is removed, tetany supervenes in about one sixth of the cases; this is the average of a series of cases reported by various surgeons, which have varied from 7 in 70 (Wölfler) to 12 in 53 (Billroth). All the patients have been young females, still in the developmental period of life. The symptoms of tetany set in during the first ten days after the operation. It does not follow partial extirpation. It has been observed in association with atrophy of the thyroid and myxœdema. This remarkable relation to thyroidectomy will be considered further in connection with the pathology of the disease.

In well-marked forms, tetany is less frequently associated with hysteria than might be expected, but hysterical contracture sometimes assumes a similar form, and it may indeed be difficult to say whether a given case is to be regarded as tetanoid hysterical contracture, or as true tetany in an hysterical subject. The acute diseases, during or after which tetany has been known to come on, are typhoid fever, scarlet fever, cholera, smallpox, rheumatic fever, measles, febricula, catarrh, and pneumonia. It has been met with very frequently in some epidemics of typhoid, usually during the latter part of the disease, after the sixteenth day, or during convalescence—in rare cases during the first week. It has also been met with in Bright's disease, and I have seen one case apparently due to lead-poisoning. Symptoms resembling those of tetany lasting a week have followed a fall on the head in a young child, which caused loss of consciousness and vomiting for two days.*

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* Cases described by Collie, Nason, Muller, Löb, and others.

Lastly, singular epidemics of tetany have been met with on the Continent. Some of these are apparently analogous to the occasional epidemics of hysterical convulsion. For instance, in a girls' school in France, in 1876, an epidemic occurred in which no less than thirty girls were attacked.† A still more singular epidemic occurred in 1846 in certain Belgian prisons. The local prevalence of the typical disease is apparently secondary to that of one of its causes, as diarrhœa in children, or typhoid fever, in which there has been, probably, some special element in the cause of the primary disease.

SYMPTOMS.—As a rule, the peculiar symptoms in the limbs come on without premonitory nervous disturbance, but in rare cases they have been preceded by headache, pain in the spine, and malaise, and even vomiting not due to gastric derangement. The muscular spasm may be the first symptom, but is usually preceded for a few hours or days by sensory disturbance in the extremities, as "tingling" or "burning" sensations. The spasm almost always sets in suddenly in the hands, occasionally in both hands and feet at the same time, rarely in the hands only, and still more rarely in the trunk. The hands feel stiff and cramped, and then become fixed. The usual posture is with the fingers flexed at the metacarpo-phalangeal joints, extended at the others; the thumb is adducted and in contact with the index finger, or is flexed beneath it, while the spasm in the thenar and hypothenar muscles renders the palm hollow. The fingers are sometimes pressed together, sometimes inclined towards the ulnar side. The position of the fingers is mainly due to spasm in the interossei muscles. The wrist is usually slightly flexed. The elbow may be free, but is more often in slight flexion. The shoulder muscles are rarely involved, but occasionally there is spasm in them adducting the arm. In a few cases the spasm in the hand muscles has been chiefly in the long flexor of the fingers, which are then flexed at all joints, and the fist thus closed. Still more rarely the wrist has been extended, and in two recorded cases the fingers were extended at all joints.

The feet are extended at the ankle-joint, and are inverted, being in the position of talipes equino-varus. The toes are strongly flexed; the knees are usually extended, rarely flexed. The thighs are sometimes adducted, rarely flexed.

In cases of slight or moderate severity the contracture is limited to the hands and feet, not unfrequently to the hands. It is very rarely unilateral. In severe cases the muscles of the trunk and head share in the spasm. The abdominal muscles become rigid, and there may be spasmodic retention of urine. Those of the back are less affected, but occasionally there is a slight degree of opisthotonos. The thorax may be fixed by spasm, which may even involve the diaphragm, and cause some difficulty in breathing, cyanosis, and even transient loss of consciousness. Sometimes the sterno-mastoids are rigid, and the head may be bent forwards. More frequently the jaws are closed by spasm in the masseters, the angles of the mouth are drawn out, and the eyelids are half closed. Even the eyeball muscles may be involved, causing strabismus, convergent or divergent, and immobility of the pupils has been observed. The tongue is sometimes stiff, and articulation may be rendered difficult by this or by the involvement of the larynx, and swallowing by spasm in the pharynx. The affection of the muscles of the face is usually only seen in severe attacks, but has been occasionally met with in attacks which were very slight.

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* Demme, 1889.

† Simon; Mattraits, 'Thèse de Paris,' 1877.

The tonic spasm may render the muscles firm and hard, and fibrillary contractions are sometimes visible in them. Rarely, paroxysms have begun by brief clonic spasm. Attempts to extend the muscles usually cause much pain. Movement is interfered with by the spasm and in proportion to it, but there is no actual paralysis. When the contracture is slight, movements are merely rendered difficult and awkward; when it is considerable, all voluntary movement may be, for the time, impossible.

Slight contracture may be painless, but when the spasm is considerable there is severe cramp-like pain in the muscles, and sharp pains pass up the limbs, especially in the course of the nerves. When the spasm is continuous, the pain may be paroxysmal, probably in consequence of slight exacerbations of the spasm. The subjective sensations (tingling, &c.) which precede the spasm may persist during the intervals, and may be accompanied by actual diminution of sensibility to touch or pain, or to both.

The spasm is usually paroxysmal. After continuing for a time, which varies from a few minutes to a few hours, and rarely to a few days, the contracture gradually passes off, to recur after a variable interval of hours or days. A feeling of stiffness often succeeds it. In some cases the spasm continues in slighter degree in the arms, rarely in the legs only, in the intervals between the paroxysms. In other cases, again, in both children and adults, the spasm may continue in moderate degree for several days, without exacerbations or with merely an occasional trifling increase. Attacks of spasm may occur and even commence during sleep, and if there is continuous contracture this may persist during sleep, although usually in less degree. Abercrombie observed no change in the continuous spasm during an attack of measles which ended fatally. Attacks are sometimes excited by muscular exertion. When due to dilatation of the stomach, percussion of the abdomen has excited attacks, and this was also observed in a case apparently due to perforation-peritonitis.

In the intervals between the attacks of spasm there is usually a remarkable increase in the excitability of the nerves and muscles in the parts in which the spasm occurs or is most intense. Percussion of the muscles, and especially of the nerves, causes a conspicuous contraction. This phenomenon may be well observed in the face, where a tap on the nerve will often cause a momentary contraction in all the muscles supplied by it (the "facial phenomenon"). It is also marked in the limbs, in which an attack of characteristic local spasm may usually be induced by compression of the artery and nerves of the limb, sometimes called, from its discoverer, "Trousseau's phenomenon." The spasm comes on a few minutes after the commencement of the pressure. It is probable that the effect is chiefly produced through the nerves, although it is said that sometimes compression of the artery is effective when that of the nerve is not; but slighter pressure on the nerve has sometimes been the most effective, and can hardly be avoided when the artery is compressed. Sometimes this phenomenon cannot be obtained. Abercrombie found the increased irritability invariably present in the face, and not in the limbs, in cases of continuous tetany of young children. Cutaneous reflex action is also greater than normal, especially on the trunk. The knee-jerk may be normal, but sometimes it cannot be obtained, probably by reason of slight persistent muscular contraction, and not because it is really absent.

There is also a greatly increased electrical excitability of the nerves of the most affected parts to both faradism and voltaism. I have obtained contraction in the muscles of the face by applying to the nerve the voltaic current from a single cell. The mode of reaction to voltaism is changed, as Erb has pointed out. The earliest contraction occurs with the positive pole when the circuit is closed, and sometimes when it is opened, instead of at the negative pole when the circuit is closed. Moreover, the closing and opening of the circuit causes not only a momentary contraction, as in health, but a prolonged contraction, "tetanus," and this with the positive pole (anode) as well as with the negative (kathode). It is the only condition in which anodal opening tetanus has been observed in man. Thus, instead of the normal reaction (see vol. i, p. 54) 1, KClC; 2, AClC, KOC; 3, AOC, we have 1, AClC; 2, AOC; 3, KClC, or 1, AOC; 2, AClC, KClC, AOTe.[*] A similar increased irritability has been found in the sensory nerves;[†] a sensation has also been produced more readily by the anode. It is said also to exist to mechanical stimulation. The increased irritability, electrical and mechanical, is greatest at the height of the disease, lessens with it, but usually persists for two or three weeks after the spasms have ceased and after pressure has ceased to be effective.[‡]

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[*] It is said that excitability of the nerve sometimes exists to voltaism only, but it is probable that in such cases it would have been found equally distinct to the separate faradic shocks, had these been used instead of the serial "current" (see vol. i, p. 376).

[†] Hoffmann, 1888; Chovstek, 1890.

[‡] It is said that the increased irritability may be sometimes found in rickety children and in weakly nervous adults, who have no attacks of spasm.

During severe paroxysms there is often copious perspiration, and sometimes local vaso-motor disturbance, redness, and even slight œdema in the affected parts; the pulse is quickened, and in some cases and in some outbreaks a rise in temperature has been observed, rarely exceeding 101° F. In other cases the temperature has been normal. The urine is usually unchanged, but transient albuminuria has been twice observed (Kussmaul, Nönchen), and in one case glycosuria coincided with the attack (Stich). Tetany has been followed by general muscular atrophy, and also by local atrophy of the thenar muscles spreading to the forearm, and accompanied by slight atrophy in one leg; the electrical irritability was lowered in the atrophied muscles.* Other trophic changes have been occasionally observed, chiefly in the growth of the nails of the feet and hands; thus necrosis has even been met with. Brownish pigmentation of the hands and face has been seen.

Varieties.—We may distinguish three varieties of tetany, according as the spasm is intermittent, remittent, or continuous, but cases are met with that present every gradation between these varieties. Although the spasm is almost always symmetrical, I have once seen it so much greater on one hand than on the other, that when slight in degree, it was one-sided. There is little relation between the course and severity of the spasm except that in the continuous form the spasm is never so violent as it sometimes is in the paroxysmal varieties. In young children tetany is usually continuous, but the intermittent form is not unknown. In older children and adults, the continuous form is less common than that in which the spasms remit or intermit.

It has been said that not only does continuous tetany persist during sleep, but that attacks of spasm may occur even for the first time during sleep. The continuous form usually, however, lessens or ceases during sleep, to return or increase on waking. It is removed for the time by chloroform narcosis.

Slight tetanoid symptoms are, indeed, not rare during sleep, especially in adult women who are in somewhat feeble general health. The patient is waked up by tingling in both hands, accompanied by a feeling of stiffness, and sometimes by actual rigidity, which may prevent all movement and last for a few minutes or longer, even for an hour or more. The fingers may be in the position characteristic of tetany, or may be flexed at all joints, but never strongly. The tingling usually lasts longer than the spasm. The condition is bilateral, and comes on irrespective of posture.† It seems to be a slight "sleep tetany." Occasionally the patients who suffer from this nocturnal tetany have similar attacks in the daytime. Now and then the attack is accompanied by some pain in the head.

Course.—In both the continuous and intermitting form of tetany, the total duration of the disease varies much. Cases of either variety may last only a few days; but the continuous form very seldom presents the prolonged duration which is not uncommon when the spasm is intermittent. If the spasm presents no considerable remission it is rare for the disease to last more than a few weeks, whereas in the cases of intermittent tetany the disease often lasts for several months. In the severe cases after excision of the thyroid, which are not fatal, the duration is sometimes prolonged; the symptoms may even continue for two or three years after the operation.

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* Weiss, 'Wien. Allg. med. Zeit.,' 1885, No. 31.

† It must not be confounded with the tingling in the region of the ulnar nerve that is due to long flexion of the elbow, or in all the fingers that is caused by pressure on the branchial plexus; in these cases the tingling is always one-sided.

Tetany is an affection prone to return on a recurrence of its exciting cause, and sometimes without appreciable cause. A mother suffered from it while suckling each of five successive children (Maccall), and a woman had an attack in each of ten successive winters (Lussana). I have also seen a case in which a woman became subject to attacks at the age of twenty-two, and was still liable to them at thirty-four; each winter, as the cold weather came on, she suffered from rheumatic pains in the limbs, and these were followed by attacks of tetany, each lasting from half an hour to one or even two days, and recurring about twice a week, until the late spring, and then they ceased until the winter. Another patient had frequent attacks, each lasting about six hours, during six weeks every spring, from the age of fourteen to twenty years.

Termination.—Most cases of tetany end in recovery. Very rarely some weakness of the legs has persisted for a time. When death has occurred, it has been usually due to the cause of the tetany, *e. g.* to diarrhœa. In a few instances, the spasms have caused death, either directly, by their violence producing fatal exhaustion (Trousseau), or indirectly, by the repeated interference with respiration causing congestion of the lungs, and a low form of pneumonia. The disease, when it follows excision of the thyroid, is much more fatal than it is under other circumstances, except when due to dilatation of the stomach which lessens the strength of the patient. Tetany has been known to be immediately followed by chorea (Salomonsen); I have met with chorea a year after tetany. As might be expected, an affection which sometimes occurs in hysteria, is occasionally associated with other symptoms of the latter disease. In one patient attacks of tetany were preceded by hysterical trismus. We have seen that muscular atrophy may supervene, either slight and confined to the hands, or general and severe. Epileptic attacks have been observed in association with tetany, and very rarely mental disturbance has accompanied it.*

PATHOLOGICAL ANATOMY.—In the few cases in which a post-mortem examination has been made, no change has been found which we can regard as the cause of the disease. The minute alterations that have been detected in the spinal cord in some cases[*] are probably secondary to its functional over-action. Such, for instance, are capillary hæmorrhages, accumulation of lymphoid cells around blood-vessels, and even minute foci of myelitis. The ganglion-cells have been found somewhat shrunken in aspect, and to contain vacuoles.[†] Hyperæmia of the upper part of the cord has been observed in some cases, but was absent in others. Softening in the cervical region, met with by Trousseau, probably indicated organic disease causing symptoms that simulated tetany, such as may be produced by a concussion of the head.

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* Frankl-Hochwart, 'Jahrbuch f. Psych.,' ix (three cases).

PATHOLOGY.—The great increase in the excitability of the motor nerves shows that there is an alteration in the function (and doubtless also in the nutrition) of the motor nerve-fibres. We know that changes in the nutrition of these fibres are usually dependent on, and secondary to, a similar change in motor cells of the spinal cord, of which the axis-cylinders of these nerves are the prolonged processes. The discovery of slight changes in the cells in severe cases, and the fact that slight spinal weakness may follow tetany, also suggest a disturbance of function of the cord, while the bilateral symmetry of the disease, and the peculiar and uniform character of the spasm, constitute strong evidence of its central origin. The sensory symptoms indicate that the disturbance of function is not confined to motor structures. Beyond this we are not at present justified in going. There is in this (as Weiss has pointed out) nothing inconsistent with the possibility that the primary disturbance may be still higher up in the brain, and that the derangement of the cord and nerves may be secondary, although, at present, the only evidence of implication of the cerebrum is afforded by the associated symptoms of insanity, hysteria, and epilepsy. These do not, however, show that the special symptoms have a cerebral origin, although it may be noted that the peculiar form of spasm ("interosseal" flexion of the fingers) is seen in some forms of spasm of brain origin as in many cases of epilepsy.

Whatever the seat of the disease, the morbid process must, as a rule, be limited to disturbance of function and nutrition, and the occasional origin of the disease in reflex irritation suggests that the disturbance of function may be, in such cases at least, the primary change. The resemblance of the spasm to that seen in ergotism has struck many observers,[‡] and has suggested its dependence on some toxic influence, possibly in dilatation of the stomach, an "auto-toxic" process, by some product of imperfect digestion or of altered secretion. The search for such an agent in the contents of the stomach has hitherto been unsuccessful. The effect of cold may possibly be due to a blood change, such as causes rheumatic fever; its action on the nerves of the skin may co-operate with a predisposition. Some etiological facts are scarcely compatible with a toxic cause. Infantile tetany has doubtless the same cause as infantile convulsions (q. v.).

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[*] By Weiss, Langhans, Ferrario, &c.

[†] The significance of the vacuoles in nerve-cells has been much discussed. I believe that they form during the process of hardening, but only when the cells are in an abnormal state.

[‡] Imbert-Goubeyre (1844), Hasse, Moxon, Eulenburg.

The frequent occurrence of the disease after excision of the thyroid is a pathological fact of great importance, but not yet of clear significance, although most observers consider that a toxic mechanism is the probable explanation. It can be produced also in animals, and follows ligature of all the blood-vessels of the organ, as well as its excision. The remarkable fact has been ascertained that if one half of the thyroid is first removed (a proceeding that does not cause tetany), and is placed in some other part, as the peritoneum, the second half can be afterwards removed, in some cases, without the result that would follow the simultaneous excision of both halves.* The malady has been thought to be the first result of the process which leads to myxœdema and the "cachexia strumipriva," and to be due to the accumulation in the blood of some material, as mucin, which it is the function of the thyroid to change. With the admission of the possibility of this we must, at present, be content.

On the whole, our knowledge of the pathology of the disease points to the nerve-cells of the spinal cord and medulla as the parts immediately deranged; whether these structures are affected primarily, or in consequence of a deranged action of the motor-cells of the cortex, we have yet to ascertain. The nerve-fibres seem to share the disturbance, almost from the first. Its definite and uniform features suggest that the derangement is a primary one of the cells themselves, and is not produced by the agency of any vaso-motor mechanism.

DIAGNOSIS.—The peculiar character of the spasm, its limitation to certain groups of muscles, its commencement in the extremities, and its bilateral symmetry, are usually sufficient indications of the nature of the case. If present, the intermissions in the spasm, the increased irritability of the nerves, and the excitation of the spasm by their compression, corroborate the diagnosis, but these symptoms may be absent in early cases. The presence of one of the most common etiological conditions (exposure to cold, diarrhœa, lactation or pregnancy), is also, in some cases, of diagnostic value. It is important, however, to remember that in slight cases the sensory symptoms may attract more attention than the motor spasm. Whenever chronic tingling in the hands and feet is complained of, careful inquiry should be made for attacks of spasm.

From tetanus the disease is distinguished by the commonly intermitting character of the spasm, by its commencement in the extremities, by the peculiar posture of the hands, and by the fact that the earliest symptom in tetanus, spasm in the masseters, is the latest in tetany. In organic brain disease, contracture is usually permanent and often unilateral; there is paralysis in addition to spasm, and cerebral symptoms are usually present. Hysterical contracture may closely resemble tetany, so far as the form of spasm is concerned, but is almost invariably unilateral, while tetany never is. When such contracture is bilateral, the case should be regarded as one of hysterical tetany. In the tonic form of epilepsy, bilateral spasm may exist, almost identical with tetany in its character, but distinguished by the extreme brevity of the paroxysms, and by the occurrence of loss of consciousness.

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* Eiselsberg, 'Wien klin. Wochensch.,' 1890. Tetany was sometimes prevented by placing the whole organ in the peritoneum, and occurred when this was afterwards removed ('Neur. Cent.,' 1892).

PROGNOSIS.—Tetany involves little danger to life except in the cases after excision of the thyroid, and those due to dilatation of the stomach. The chief danger is from the cause of the tetany, such as severe diarrhœa. It is only when the paroxysms are very violent, causing considerable and repeated interference with respiration, with signs of œdema of the lungs, that there is any ground for apprehension. The duration of the disease is difficult to foretell. When the attacks are prolonged, lasting several days, the affection is not likely to be of long duration, and continuous tetany, of slight intensity, will certainly soon be over. Prompt treatment of a commencing case will frequently cut it short. As long as the irritability of the nerves is increased, the paroxysms are likely to recur. It must not be forgotten that the subjects of tetany are very liable to future attacks, if exposed to an adequate cause. The tetany of pregnancy, it must also be remembered, usually continues until delivery.

TREATMENT.—Whenever the cause of the disease can be traced, the removal of this or the mitigation of its general effect, is the first and most important element in treatment. If resulting from exposure to cold, and seen early, warm baths, or free diaphoresis, should be employed. Pilocarpine has been used with advantage, but is seldom necessary. The state of the bowels must be carefully regulated. Lactation should be stopped, and strength restored by tonics, especially by iron and quinine.

In most cases the spasm may be relieved most effectually by full doses of bromide of potassium, ʒss three times a day. Chloral, Indian hemp, and hypodermic injections of morphia have also been found useful for the same purpose. The inhalation of chloroform at first increases the spasm, but all contracture usually disappears when the patient is fully under its influence; at the same time chloroform has little influence in preventing a recurrence. Two cases, however (recorded by Hauber), which had resisted other treatment were quickly cured by a course of chloroform inhalation, with energetic massage during the narcosis. The local application of stimulating liniments (especially chloroform) and of cold to the extremities, also sometimes arrests the spasm. Trousseau observed benefit from the application of ice to the spine. Electrical treatment has been followed by improvement in some cases, and has failed in others. The best results have been obtained with the voltaic current, the negative pole to the spine, the positive on the irritable nerves. Faradism is contra-indicated.

In France, tetany was once invariably treated by bleeding; the symptoms rapidly disappeared in some cases, especially in men, occasionally also in weakly women. The procedure often failed, however, when tonics quickly cured the patient. As a rule, when the spasm has ceased, nervine tonics are desirable, as strychnia, quinine, arsenic, iron, or valerianate of zinc. I have found the two last, with bromide, of great service in chronic paroxysmal tetany.

The treatment of tetany in children is, in the main, similar to that of their convulsions. Attention to the underlying diathesis is of paramount importance. In young children, symptoms of rickets are rarely absent, and cod-liver oil and iron are the most effectual remedies, the continuous tetany of early life being less amenable to sedatives than the intermittent tetany of older children and adults. Cheadle found bromide and chloral useless in one case, in which the extract of Calabar bean succeeded, in doses of one thirty-sixth of a grain increased gradually to a third, but cod-liver oil and iron were given at the same time. In addition to the latter, the administration of bromide should never be omitted.

For nocturnal tetany I have found no remedy so useful as a dose of digitalis given at bedtime. Bromide often removes the symptom, but occasionally fails. Tonics may also be given with advantage during the day.

TETANOID CHOREA.

A case was recently under my care that presented symptoms intermediate between those of chorea and of tetany. The disease was fatal and no lesion was found after death. The patient was a boy aged ten. A brother was said to have died from some affection similar to that from which this child was suffering. There was a history of three other relations having suffered from maladies resembling chorea. In the patient the symptoms commenced gradually, seven months before death. They consisted of tonic spasm, which was continuous, and varied by paroxysmal attacks of similar, but more intense, spasm. The face was involved on both sides, so as to cause a constant peculiar smile. The tongue was pressed back against the palate in such a manner as to impede swallowing and prevent speech. The arms were extended, pronated, and rotated inwards, so as to bring the back of the forearm outwards, while the fingers were generally slightly flexed at all joints, but at times were extended, and slowly moved in the irregular way characteristic of athetosis. The legs were extended at all the joints, the feet being over-extended in talipes equino-varus, and the toes were flexed. At times the spasm at the hip became flexor, so that the extended legs were raised off the bed. The muscles of the trunk were also involved in the spasm. At first the left side was the more severely affected, but afterwards the spasm became equal on the two sides. The electric irritability of the muscles was normal, and there was no mechanical excitability of the nerves. There was considerable pyrexia during the more severe stage of the disease. The boy steadily emaciated, and died from exhaustion. The whole central nervous system appeared normal to the naked eye, and no distinct morbid appearances could be discovered on microscopical examination. I have not been able to find a description of any quite similar case.

OCCUPATION NEUROSES.

The term "occupation neuroses," adopted from the German ("Beschäftigungs-neurosen"), is a convenient designation for a group of maladies in which certain symptoms are excited by the attempt to perform some often-repeated muscular action, commonly one that is involved in the occupation of the sufferer. The symptoms occur in the part by which the action is effected, and the action is interfered with by them. Other acts (at least in the early stage of the affection) do not excite the symptoms, and are not interfered with. The most frequent symptom is spasm in the part, which disturbs or prevents the due performance of the intended action, and hence the word "cramp" is commonly applied to it, qualified by the name of the action, or occupation, that excites the disorder, as "writers' cramp," "pianoforte-players' cramp," "telegraphists' cramp," "sempstresses' cramp." The term "cramp" sets forth the muscular spasm which, in most cases, is the chief, but rarely the sole symptom. There is usually not only spasm but pain, which is not the direct result of the spasm, and may be the only obtrusive symptom. It may be referred to the muscles, or to the bones or joints, or to the position of certain nerves. In the latter case, it resembles neuralgia, and the more closely, since certain foci of pain and tender points may be developed, as in ordinary neuralgia, from which it differs in being at first not spontaneous, but excited only by the special action. Hence we must distinguish a motor and a sensory form, a spasmodic and a neuralgic variety of occupation-neurosis, but the two forms are very often combined. Although the disturbance is in the first instance confined to the special action, and may remain limited to this, it often extends, after a time, to other actions in varying degree, and in proportion as the muscular contractions correspond, in combination and character, to those of the movement first disordered. The extension may ultimately be so wide, that spasm, and still more often pain, occurs on any movement. I have even met with cases in which a spasm, at first excited only by a special act, ultimately extended not only to all acts, but occurred spontaneously. Thus, in their ultimate extension, the occupation-neuroses are not so well defined as in their earlier stages. Moreover, they are not always well defined in their causation. Local disease or injury, or some affection of the central nervous system, weakening the limb, may distinctly co-operate in developing the morbid state. In such cases, extreme care is necessary in determining the nature of the case, because a local affection is often first *revealed* by the interference with a delicate action, performed frequently and for a long time. Many forms of disease are thus erroneously supposed to be of the nature of occupation neuroses, when the occupation merely manifests and does not cause the symptoms.

The most common form of occupation-neurosis is that of writing, and since this is not only the most common but is typical of the class, it will be most convenient to describe it at length, the conclusions regarding it being applicable to all other forms.

WRITING NEUROSIS; WRITERS' CRAMP.

Writers' cramp was first described by Sir Charles Bell in the year 1830. It was called "Scriveners' Palsy" by Solly, a term still sometimes applied to it.[*]

ETIOLOGY.—The affection is very much more common in males than in females, especially in its motor form, doubtless because fewer women are engaged in occupations that involve a large amount of writing. If all forms are included, males constitute two thirds of the sufferers, but a still larger proportion, at least five sixths, of those who present the motor form. It is a disease of the active period of adult life, rarely commencing under twenty, not often after fifty, while about five sixths of the cases begin during the intervening thirty years. The distribution of 153 cases[†] is as follows:

	10–		20–		30–		40–		50–		60–
Cases . .	2	...	52	...	50	...	32	...	12	...	5

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[*] The most important writings on the disease are those of Berger ('Eulenburg's Real-Encyclopædie,' art. "Beschaftigungs-neurosen") and of Poore ('Practitioner,' 1872-3; 'Text-Book of Electricity,' and 'Med.-Chir. Trans.,' vol. lxi). The last article contains a large number of carefully observed facts.

[†] 33 cases described by Poore ('Practitioner,' 1878), 64 collected by Berger (loc. cit.), and 54 cases of my own; all being rejected in which, from the character of the symptoms, there was any room for doubt as to the nature of the case.

Thus about one third of the cases commence in each of the third and fourth decades of life, and only a fifth in the following ten years.

An hereditary tendency to nervous affections can often be traced. One of my patients had an epileptic uncle; another lost a brother from general paralysis of the insane; three others had each an insane parent, and one an epileptic father. In some instances, more than one member of a family has suffered from the special affection. Vance relates the case of a patient who, to rest his hand, went on a visit to a distant brother, and was surprised to find that he also had given up writing from the same cause. I have met with a very characteristic case in a lady whose father also suffered from writers' cramp. In estimating a family tendency, it must be remembered that writers' cramp is a disease that is easily imagined, especially by those who have witnessed the disorder. The malady is sometimes met with in those who exhibit no other symptom or tendency to nervous derangement, but more frequently the subjects of it are of distinctly "nervous" temperament, irritable, sensitive, bearing over-work and anxiety badly, and occasionally the neuropathic tendency shows itself in other independent disease. For instance, one patient with characteristic writers' cramp (spasm absolutely confined to the act of writing) subsequently presented the early stage of tabes; others had previously suffered from migraine, epilepsy, and melancholia; and many of those who suffer from pain in writing, alone or with spasm, have been liable to neuralgia elsewhere, or have presented various manifestations of nervous weakness. Two of Berger's patients were epileptic, and one of my own had some fits in early adult life. Another had suffered from infantile paralysis in one leg. We must distinguish, however, between such cases, in which an independent disease shows the existence of a neuropathic tendency, and the cases already referred to, in which a nervous affection of the part employed in writing, is one of the causes of the special disease.

Whatever lowers the general tone of the nervous system may doubtless act as a predisposing cause, but no influence is met with so frequently as to deserve special mention, except anxiety. It is remarkable how many patients, at the time of the onset of the affection, were enduring anxiety from family trouble, business worry, or weighty responsibilities. As an instance of the potent influence of anxiety I may mention the case of a clerk in a Government office who had done only a moderate amount of writing, but became affected with writers' cramp during an anxious lawsuit about his wife's property. He took six months' rest, and at the end of two months his handwriting was as good and free as ever. Towards the close of his holiday his wife became seriously ill; while nursing her, and before resuming his work, the writing cramp returned.

An example of the influence of local disease or injury was a naval officer who sprained his thumb, and before it had recovered had to do a considerable amount of writing in preparing a report of a surveying expedition; characteristic writing spasm came on. In many recorded instances some painful affection of a finger has preceded the onset, and in one case, periostitis of the external condyle of the humerus (from which some of the forearm muscles arise), distinctly co-operated in causing the cramp; pressure on the tender part excited reflex spasm (Runge). In two patients, characteristic writers' cramp followed recovery from a slight attack of right hemiplegia (Vance, Runge). Primary neuralgia and neuritis in the arm may also aid in the causation of the disease, but nerve-tenderness, which is common, is probably, in most instances, part of the affection and not its cause, and does not show that there is a primary disease of the nerves.

The chief agent in the production of the malady is the act of writing, which has usually been excessive in degree. Hence the affection occurs chiefly among those who earn their living by writing, and clerks furnish the majority of the cases. But it now and then occurs in persons who have not done an excessive amount of writing; and in such cases a powerful predisposing cause, such as anxiety or neuralgia, may commonly be traced. The occurrence of the disease is influenced less by the amount than by the manner of writing. There are, in writing, two chief elements—the way in which the pen is held, and the way in which the movements are effected. The mode in which the pen is held is comparatively unimportant; it is the mode in which the pen is moved that chiefly determines the occurrence of the disease. The movement may be effected either by the muscles moving the fingers and hand, or by the muscles of the upper arm, and according to the mode adopted does the necessary degree of muscular contraction vary. The smaller the muscles employed, the greater must be the relative degree of contraction to produce a given movement of the pen, the greater is the amount of fatigue produced, and the more readily does cramp occur. Writers' cramp is almost entirely confined to those who write in the first two of the following four modes:—(1) The worst mode of writing is with the little finger as a fixed point of support. The pen is then moved up and down by the muscles of the thumb and first two fingers, which are constantly contracted almost to their maximum; the lateral movement is effected by a slight supination of the hand; only a few letters can be written without the movement of the little finger, and before it is moved a considerable strain is necessary to prevent the pen being raised from the paper by the process of supination. Of all modes of writing, this is the worst and most common. (2) Another mode is to write from the wrist as a fixed point. In this method, the up- and down-strokes are made chiefly by the flexion and extension of the fingers and thumb in the manner above described, but the lateral movement of the hand is effected chiefly by the ulnar abductors of the wrist; as these are also flexors and extensors, considerable effort is necessary to keep the pen on the paper during the left-to-right movement. (3) A far better way is to write from the middle of the forearm, the wrist moving with the hand, and the lateral movement being effected by adduction of the humerus, the forearm rotating on its point of support as on a pivot. Even in this method, however, since the tendency of the hand is to move in a curve, effort is necessary to keep the writing in a straight line, and this has to be effected by increasing flexion of the wrist, which interferes with the action of the flexors of the fingers in moving the phalanges. (4) The best and only free method is to write from the upper arm and shoulder, with no fixation of the arm; the forearm, wrist, and little finger rest on the table so as to take off some of the weight of the limb from the shoulder-muscles, but both wrist and forearm move along the table as the writing progresses from left to right.* In this way the pen can be held very lightly; very little of the movement is effected by the small muscles of the hand; the fingers scarcely alter their position, except when a stroke is carried far above or below the line; and even for this a movement of the fingers is not always necessary. No style can be considered free unless it is easy to write a whole line across a page of foolscap without once breaking contact between the pen and the paper.

It is probable that the practice of thickening the down-strokes conduces to the occurrence of the affection, as it necessitates frequent increased pressure on the pen. Many writers have suggested that the use of steel pens is one element in the causation of the disease, and it seems probable that this is true, since steel pens have to be grasped more firmly, and adjusted more exactly, than a quill, and also because the disease seems to have become more frequent since the use of steel pens has become almost universal. But writers' cramp occurred when quills were the invariable instruments of writing, and is still seen occasionally in those who never use a steel pen.

Among clerks who suffer, lawyers' clerks constitute an undue proportion. This is no doubt due to the cramped style in which they commonly write. On the other hand, writers' cramp is practically unknown among those who write more, and under higher pressure, than any other class,—shorthand writers. The speed required, and the style needed, for forming shorthand characters, compels a very free style of writing, generally from the shoulder, and this is also adopted by them in longhand writing, and the result is that they have an almost complete immunity from the disease.† I have it is true known the cramp to occur when the patient was writing longhand, and not when he was writing shorthand. It ultimately involved the latter, but he was not a professional shorthand writer, and his longhand method of writing was bad.

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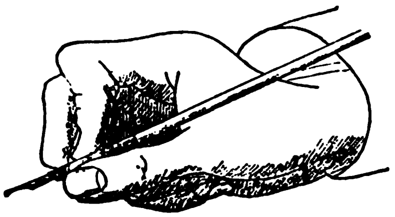
* To some who are accustomed to write with the fingers chiefly, the statement in the text may seem incredible, but I have verified it by a pencil fixed to the forearm, which produced the same characters as were traced by the pen held in the hand.

† I have indeed seen one case in a shorthand writer, but it was not quite a pure case, and at the meeting of the Shorthand Congress I could not hear of any stenographer who had known of a case of writers' cramp in a member of his profession.

SYMPTOMS.—The precise mode in which the act of writing is deranged varies much in different cases. The chief disturbance is usually from spasm, but sometimes it is from pain. Occasionally there is tremor without other spasm, ceasing when the attempt to write is relinquished. More rarely still there is simple inability to write, apparently from weakness, without spasm, although there is no loss of power for other movements. Four forms may thus be distinguished, the spastic, tremulous, neuralgic, and paralytic forms, but the last is extremely rare, and I have never met with a distinct example of it.

Motor symptoms.—The spasmodic form is the most frequent and the most characteristic, but it presents considerable variety in its features, dependent partly on the mode in which the patient is accustomed to write, and the muscles which he accordingly calls into chief action. The commencement is almost always gradual (some instances of sudden onset will be mentioned presently). After writing for some time the patient finds something unusual about his writing; the pen does not move quite as he intended it to do; a stroke now and again is irregular, extends too high or too low; a slight involuntary movement causes an unintended mark. He finds that he is grasping the pen too tightly, and cannot help doing so; that the fingers do not keep in their accustomed place; and the first finger has a tendency to slip off the pen, so that this gets between the first and second finger. He endeavours to mend matters by taking a firmer hold, but this seems to increase the difficulty, and he finds that he writes slowly, as if a weight were attached to the hand. The hand feels strangely tired, and an aching pain in the finger or thumb or first metacarpal bone, or in the wrist or forearm, makes it still more difficult for him to go on writing. These symptoms may continue, with only slight impairment of the power of writing, for weeks or months, but they occur after writing for a shorter time; they increase in degree, and now and then there is distinct spasm, which cannot be controlled. The first finger or the thumb tends to become flexed at the middle joint, so that its tip moves up the pen, or, less commonly, the fingers become extended, so that the pen is not pressed against the paper with sufficient force, and may even drop from the hand, or the thumb may become extended across the pen. The characters of the writing become still more irregular, the down-strokes are too thick, the point of the pen may be driven through the paper, and in its irregularity of form and force the writing "resembles that done in a jolting carriage." Rarely the chief spasm is in the fourth finger, or in the third and fourth fingers, and pain may be felt in the long flexor of these fingers, and the ulnar flexor of the wrist. Sometimes the whole hand seems to get stiff, and its movements slow; in such cases there is a tendency for the letters that are formed to become smaller and smaller as the writing proceeds, until they are illegible. As the spasm increases in degree, it extends in range, and involves more of the muscles of the forearm. There is a tendency for the wrist to become flexed, or extended, or supinated, and in the effort to prevent the disturbing movement, the opponents contract strongly, until at last all the muscles of the forearm may be in such energetic spasm as to render movement of the pen impossible. Various devices are at first employed to counteract the spasm. The mode of holding the pen is changed; it is held between the first two fingers, or fixed in a piece of cork, which is grasped by the hand, and the movements in writing are effected by the upper arm; or the patient fixes the right hand by the help of the other; placing, for instance, some fingers of the left hand between the two last fingers of the right. For a time these devices give a little help, but the spasm gradually increases in degree, and overcomes the fixing help, or it spreads to the muscles of the upper arm.

Fig. 155.



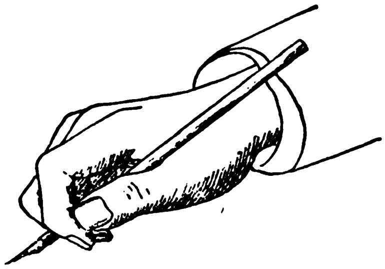


Fig. 156.

Fig. 155.—Cramped method of holding pen, habitual to a patient who suffered from writers' cramp.

Fig. 156.—Pen held between first and second fingers, by a patient suffering from difficulty in writing.

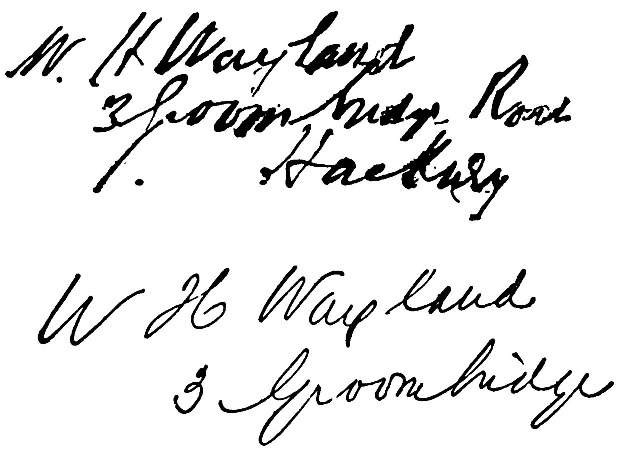


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Fig. 157.—Handwriting of a patient suffering from writers' cramp, and after recovery.

Although the onset is gradual in almost all cases, in very rare instances the affection comes on in an acute manner. In the cases of this character, pain has generally been a permanent symptom, and some slight symptoms have usually preceded the acute onset. One patient, who had noticed a slight "crampy" feeling in his fingers after writing for a long time, one day wrote rapidly for several hours, and then the hand suddenly became so stiff that he could scarcely move it. He gave up, but next day was no better, and when I saw him, ten days later, he could only write two words, and that with extreme slowness and effort, and the attempt caused much pain. Another patient, who had been conscious for some time of a little more fatigue in writing than was usual, sat down one day to write a number of letters on a subject that vexed her much. After writing for about two hours she felt a sudden pain around the wrist, passing down to the knuckles, and up the forearm to the elbow. The hand then slowly closed in spasm. She forced the fingers back, and went on writing for a short time with much pain and difficulty. Subsequently for some years, as soon as she attempted to write, the same pain came on, followed by spasm. Other acts, after a time, also excited the pain, although much less readily than writing.

The spasm is almost always tonic in character; and although it may now and then be varied by a slight start or jerk, there is very seldom actual clonic spasm. Clonic spasm is, however, sometimes met with; in one instance, as soon as the patient attempted to write, the first finger and thumb became flexed at all joints by clonic spasm, and slipped off the pen; in another, as soon as the pen was adjusted in its proper position by the help of the left hand, the fingers and thumb began to jerk, and when the pen was put against the paper, the thumb displaced the pen over or under the forefinger. A sudden jerk in one of the fingers, extending for instance the first, second, or little finger, is not uncommon, nor is a sudden jerk of the whole hand, causing an unintended stroke on the paper. Tonic spasm is often accompanied by some tremor, and occasionally the tremor is a conspicuous symptom. The letters are "shaky," and the lines are varied here and there by angular zigzags. As soon as the attempt to write is relinquished, the tremor usually ceases. When confined to the act of writing it is sometimes limited, as to the thumb. It is rare to meet with tremor only; in most cases spasm is associated with it; there may be at first simple spasm and afterwards some tremor in addition, or tremor may at first occur alone and afterwards tonic spasm as well as tremor. Occasionally, in old-standing cases, there is slight tremor in the hand when the patient is not writing, and when this is the case there is often some tremor in the left hand as well as in the right.

The spasm may be limited to the act of writing, and other actions, even such as involve delicate muscular co-ordination, may be performed without the slightest difficulty. It is not uncommon, for instance, for the patient to be able to shave himself, or to play the piano, with perfect facility. In slight cases the spasm may be limited even to the act of writing with a pen, and the patient can write with a pencil without difficulty.* I have known a patient to be able to paint without difficulty, although he could scarcely write. A still more curious limitation has been observed; printed characters could be traced with a pen, although an attempt to write in the ordinary manner at once brought on spasm.

But absolute limitation to the act of writing is seldom met with in severe cases of long duration; there is generally some difficulty in actions requiring delicate co-ordination of the same muscles. One patient had no difficulty in any other action except in shaving himself. Another, at the end of twelve years, could do everything except draw and scratch out with a penknife. The extent to which the spasm spreads to other actions varies very much in different cases. It is greatest in cases in which flexor spasm predominates. Occasionally, in such cases, the spasm comes on when any action is attempted, and I have known one case in which spasm, at first confined to the act of writing, ultimately not only extended to all other actions, but became spontaneous, so that when the hand was at rest the fingers and wrist gradually became flexed.

Power in the hand may be quite unimpaired. Sometimes the grasp is a little weaker than it should be, and it is not uncommon, as Poore has pointed out, to find definite slight weakness of certain muscles of the hand. Occasionally there is considerable loss of power, and inability to sustain effort. There may, in rare cases, be slight wasting of certain muscles, but this is altogether exceptional in cases of true writers' cramp.

The electric irritability of the nerves and muscles may be perfectly normal, or may present a slight change, increase or diminution, chiefly in cases that have lasted for some time. The change is usually the same to faradism and voltaism, and the degree of irritability is similar in both muscles and nerves. It is often found in all the nerves which are accessible to examination. It is thus to be regarded as primarily a change in nerve irritability, not of muscle irritability; the change in the irritability of the muscles depends on the motor nerve-endings they contain.† It may be remembered that all three nerves of the arm, radial (musculo-spiral), ulnar, and median, supply muscles employed in writing. Several cases that I have seen, show that increased irritability is the earlier change of the two, and that diminution of irritability succeeds it.

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* Partly because he can press more firmly on the pencil, and thus steady the hand (see Poore, 'Med.-Chir. Trans.,' vol. lxi, p. 127).

† At the same time it would be wrong to lay much weight on this consideration, because it is possible that an abnormal irritability of the muscular fibres may cause them to respond more readily to a given stimulation of the nerve-fibres. We can only judge of the stimulation of the motor nerves by the effect on the muscular fibres. It is, however, customary to regard a change in the muscular effect of stimulation of the nerves, as evidence of a change in the nerve-fibres themselves.

Sensory symptoms are seldom entirely absent, and are often very prominent. They may exist alone ("sensory form"), or may be combined with motor symptoms. Often, at first, there is only a general sense of distressing fatigue in the hand, to which spasm may be afterwards added. Definite dull pain is often referred to the bones or joints, very often to the metacarpal bones or to the wrist, soon ceasing when the effort to write is relinquished. In one case it was felt only in the middle and fourth finger, passing from the latter up the outer edge of the hand which rested on the paper. Sometimes it is accompanied by local tenderness. Occasionally, though not often, a tingling sensation, "pins and needles," is felt in certain fingers during the attempt to write. Now and then the pain is more severe, and may be referred to the course of the nerves, so as to be distinctly neuralgic in character. At first it occurs only during the act of writing, but after a time it may be produced by any muscular effort in the part. It may be chiefly localised in certain foci along the course of the nerve, and after a time the nerves are found to be tender, and the tenderness is greatest at the foci of pain, these tender points thus completing the resemblance to ordinary neuralgia. In many cases some muscular spasm accompanies the pain during the act of writing, and often has preceded the pain in time. I may briefly relate a most instructive case of this character. The patient was a young woman, a lithographic writer, producing exquisite copper-plate writing, which could only be executed in a cramped manner, with fixation by the little finger. She found that her hand grasped the pen with undue force, and now and then a stroke did not go quite in the intended direction. The spasmodic symptoms slowly increased, without the slightest pain, for three months, the pen tending to slip between the first and second finger until she was obliged to hold it thus. Then slight pain was felt in the thumb and fingers, at the places where they were in contact with the pen. This pain gradually extended to the wrist, and then up the arm to the axilla, and continued until she came under my care two years after the onset. The spasm remained about the same, but the pain was so great as alone to prevent her from writing. It still commenced where the pen was in contact with the thumb and with the contiguous sides of the first two fingers, being felt in all three, and darting up the arm. A focus of greater pain was situated in front of the wrist over the median nerve, and another a little below the elbow; these points were very tender to pressure. There was also some general tenderness of the median and ulnar nerves, and another tender point on the musculo-spiral. The pain never occurred spontaneously, but was now excited by any muscular effort. The nerves presented a distinct increase of electrical excitability. Thus the prominent trouble at the time the patient came under observation was neuralgic pain, but both the history and the character of the pain showed that it was secondary to writing and a true "occupation neuralgia." In some cases the pain may become altogether independent of the act of writing. In one patient it was associated at the onset with well-marked spasm; writing was entirely given up, and the spasm ceased, but the pain continued and was produced by any movement, and ultimately it was felt chiefly in the upper part of the arm and then in both shoulders, and was brought on by emotion at well as by movement. The sensory symptoms, when they once become prominent, often exhibit a strong tendency to spread, and sometimes acquire a relative independence, as in the case just mentioned. The feeling of "tingling" or "pins and needles" already mentioned as an occasional symptom, may also spread, like the tremor, to the other arm.

Course.—The symptoms continue, and usually increase as long as the patient perseveres in the attempt to write, and only become stationary when prudence or sheer inability prevents the effort. The rate of increase varies much in different cases, and is generally rapid in proportion to the badness of the original method of writing. The tendency for the affection to spread to other actions also varies much, and is generally proportioned to the general weakness of the nervous system. As we have seen, the sensory symptoms of neuralgic type exhibit a greater tendency to extension than do the motor symptoms. The anxiety caused by the affection, in those who depend for their living on the action deranged, often causes general nervous weakness, which, in its turn, tends to increase the malady. It is not common to have actual recovery and later recurrence; one patient had typical cramp for eighteen months at thirty-three, which passed away with rest, to return at forty-one.

The sufferer who finds himself unable to write with one hand often learns to write with the other. After he has acquired the needful facility, and has written with the left hand for a time, similar symptoms may develop in this hand, and they then usually progress more quickly than in the arm first affected. Sometimes, even when the left hand does not suffer, the action of writing with it causes synergic movements of the right hand. When sensory symptoms are prominent, the left hand is especially apt to suffer early. Fortunately the invasion of the left hand is not invariable; I have known it to do good work for twelve years, although the affection continued unchanged in the right hand. Indeed, if we exclude the cases of neuralgic type, I think that the chances are nearly equal whether the left hand will suffer or not. If perfect rest is given to the right hand, either by abstinence from writing or by the exclusive use of the left hand, the symptoms may slowly lessen, and ultimately pass away, so that writing may be resumed, without any return of the affection. This was the case with the patient whose handwriting is shown in Fig. 157. A more typical case of writers' cramp could not be, and yet the recovery, during two years' use of the left hand, was so complete that he has now written, and written much, with the right hand for thirteen years, without any recurrence of the disease. In some cases, however, the symptoms that have been removed by rest return soon after the attempt to write is made, especially if the period of rest then given to the right hand is too short; in others, even rest fails to effect much improvement, and the symptoms may continue to the end of life. The tendency of neuralgic pain to spread widely has been already mentioned. When tingling is felt in the fingers, it is occasionally also felt in the foot of the same side, and I have once known slight cramp in the leg to accompany such tingling and severe spasm in the hand.

PATHOLOGY.—No anatomical changes are known in writers' cramp, and none could be found in one case which I had an opportunity of examining. The pathology of the disease has therefore to be inferred from its symptoms and causation. Three chief theories have been held regarding its nature. According to one, it is essentially a local disease; a weakness in some muscles permits the over-action of their antagonists, which increases to spasm.* A second theory ascribes the spasm to reflex action, the result of the stimulation of the sensory nerves in the act of writing.† A third (held by most writers on the subject) regards the affection as primarily and essentially central, the result of a deranged action in the centres concerned in the act of writing. The first two theories seem inadequate, by themselves, to explain the symptoms. Most observers have failed to find, in the early stage of true writers' cramp, any evidence of primary weakness. The initial disturbance is spasm; the slight loss of power which may be found in certain muscles after the disease has existed for some time does not explain the spasm, first because this is far wider in range than the antagonists of the feeble muscle; secondly, because the very muscles that are weak are often involved in the spasm; thirdly, because local muscular weakness does not excite spasm of the character presented by a well-marked case of writers' cramp. The reflex theory is inadmissible because, in the majority of cases, there is no evidence that abnormal sensory impressions precede the spasm. Even in the case related on p. 719, in which pain was so prominent, it was distinctly secondary in time. Both weakness and pain in the hand are very common without the manifestation of the slightest tendency to the occurrence of writers' cramp. Although neither the theory of local weakness, nor that of reflex spasm, can be regarded as explaining the phenomena of ordinary cases, it is very probable that each influence may take a share, in some instances, in increasing the disease, and occasionally may help to excite it. But the morbid state of the centre may be developed without such aid, and must be regarded as the chief element in the disease.

All forms of occupation-spasm affect movements that are purely acquired, and of all these movements that of writing is the most complex and most delicate. The acquisition of the power of performing any action with ease, means the "education" of the nervous centres concerned in it, the establishment of a tendency to the associated action of nerve-cells, in perfect adjustment of varied order and degree, with the least possible voluntary effort of excitation and control. This, in the language of modern physiology, means the establishment of lines of lessened resistance in centres, *i. e.* between connected cells, and doubtless also modifications in the force and readiness with which nerve-energy is liberated in them. This education is effected by the repeated functional action of the centres under the stimulus of the will, at first strong, afterwards gradually lessening, as the desired functional mechanism is developed by exercise, and established by the resulting modifications of nutrition. There, in health, the process stops. Readily as nerve-energy is evolved, it is always under a voluntary stimulus, and exactly in the degree required. The clearest conception we can form of the pathology of writers' cramp is that this process of lowering of resistance between nerve-cells has gone too far, so that the energy evolved is excessive and irregular, although, when the same cells are excited in a different order, the resistance is normal in proportion as the order differs from that involved in the act of writing, and hence, in that degree, other movements escape. No doubt the impairment of function is attended by impairment of nutrition. In some cases the changes go so far that all attempts at movement give rise to cramp, and, at the same time, the total power that can be exerted is considerably below the normal.

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* Dzondi, Meyer, Haupt, Zaradelli. This theory has been ably advocated by Poore.

† Fritz, Romberg.

The mechanism of the morbid lowering of resistance may be the same as that by which the resistance was lowered in the education of the centre, carried in this case to excess. The degree of exertion necessary to produce the effect varies inversely as other influences co-operate, and tend to degrade nutrition or to disturb the centre. Some causes, such as anxiety, aid in the former way, by impairing nutrition; others, such as local disease of the limb, in the latter, by deranging the centre. The influence of local pain and local weakness in deranging movement is too familiar to need illustration, but this effect is probably produced through the centre, and can only aid in the production of writers' cramp by disturbing the normal relation of the force evolved, and so aiding in the establishment of the morbid state just described.

The condition assumed is that which used to be described as "irritable weakness." In all parts of the nervous system, when force is too readily evolved, the total amount of nerve-force which can be liberated is usually below the normal. Thus there may be weakness as well as spasm, and as the morbid condition may progress in one part of the centre more than another, the weakness may be distinct only in certain muscles. The fact that the affection occurs chiefly in those who write by means of small muscles, is quite compatible with the theory that the disease is essentially central. The difference between the method of writing by small or by large muscles is that in the one case there is extreme activity of comparatively few cells of the cerebral cortex, in the other case there is slight activity of many cells. The morbid state of nutrition which accompanies disturbed function in a motor centre tends to descend the motor fibres, and hence we can understand that a slight change in the electric irritability of the nerves should sometimes be found, just as in chorea.

It has been objected to the central theory that it assumes the existence of a centre for the co-ordination of writing, and therefore of every separate action which any one part of the body can perform, and that we know nothing of the existence of such centres. The objection is invalid because no such assumption is made. The theory does not assume that there is a separate centre for writing or other movement, but only that there are varying lines of resistance in the innumerable connections of the motor nerve-cells, and that, as each movement is the result of the action of nerve-cells in different order and degree, permanent functional relations are developed and may be separately deranged. No separate co-ordinating centre is assumed, any more than in a complex piece of machinery, in which the bars and bands and wheels that convey the force co-ordinate it by their arrangement. It must also be remembered that the conception of a physiological centre does not necessarily involve that of a local limitation. Nerve-cells act together that are far apart, and those that are adjacent are often independent. Diffused through a mass of grey matter may be many separate mechanisms, not necessarily more in one part than in another, and if we call them "centres" we must not allow mathematical conceptions to govern our physiological ideas.

The tendency, often seen, for the other hand to be affected, affords additional evidence that the disease is essentially central. Many facts show that there is a close functional connection between the similar mechanisms on the two sides. Thus, as already mentioned, a patient, when he tried to write with his left hand, found his right fingers performing slow movements of flexion and extension. On any theory, it is difficult to say why the second side should suffer in some cases and not in others.

One other symptom remains to be discussed—the neuralgic pain, which gives to some cases a special character. The act of writing involves afferent impulses and sensory impressions, unnoticed till they become increased by fatigue. When cramp occurs, these become excessive. All increased sensation means increased action of the sensory centres, and this, once established, may assume a morbid independence, as in many cases of ordinary neuralgia (*q. v.*), and, as also in neuralgia, nerve-tenderness may be secondarily developed. There is no justification for regarding these tender points as evidence of neuritis, for even in such a case as that recorded on p. 719 the symptoms are merely such as are met with in many cases of neuralgia of primarily central origin. This case is especially instructive as showing how the pain, *i.e.* the morbid action of the sensory centre, became gradually developed from the increased afferent impressions caused by the spasm. The pain started from the points at which the nerves of the skin were unduly stimulated by too firm a grasp of the pen. It is scarcely necessary to point out that the same predisposing influences which facilitate the occurrence of that state of motor centres which shows itself in spasm, may also conduce to the development of a like state in a sensory centre, felt as pain. But structural changes in the nerves may occur as a result of pain that is purely central in origin, and the motor nerves, in central diseases, as chorea and paralysis agitans, may present alterations in electrical excitability, which are evidence of secondary changes in their nutrition. It is not surprising that such changes should be found in "occupation neuroses," even assuming their central origin. Such changes, once set up, may develop in relative independence, especially under the reflex influence of pain, and may even induce secondary changes in the irritability and nutrition of the muscles. These are exceptional, but they are open to the misinterpretation of being regarded as the primary element in the disorder.

There is, at present, no direct evidence as to the part of the nervous system in which the primary derangement occurs. That the action of the motor and sensory nerve-cells of the spinal cord must be disordered is certain, since it is through them that spasm is produced and pain perceived. But we do not know whether their derangement is primary, or whether it is simply the effect of a primary disorder in those cerebral centres in which the movements are arranged. The latter is the more probable, and the central region of the cortex is that to which we should naturally turn as the seat of the primary derangement, since it is from this part of the brain that the spinal cells are directly excited. Analogy suggests that the sensory disturbance is spinal in a larger degree than are the motor symptoms.

DIAGNOSIS.—A well-marked case of writers' neurosis is rarely mistaken for any other affection. The initial limitation of the symptoms, whether spasm or pain, to the act of writing, sufficiently indicates the nature of the case. But the converse error is often made; cases are supposed to be examples of writers' cramp which are of a different nature. Many paralytic and painful affections of the hand are discovered by the interference they cause with the action of writing, because it is the most delicate action that the hand is called on to perform, and they are supposed to be specially connected with the act, when this merely reveals their existence. I have several times known the mistake to be made in hemiplegia of gradual onset, and also in a case of commencing insular sclerosis, and in another of early tabes affecting the arms. Even pressure-palsy of the musculo-spiral nerve has been ascribed to writing. Still more frequent is the error in cases of pain in the arm of various kinds. In acute neuralgia and neuralgic rheumatism of the forearm muscles, writing increases the pain, and is interfered with by it in a way that renders the mistake easy. It is needless to enumerate the various other affections that may be thus confused, because the simple rule above given suffices for their distinction—from the first, the symptoms are not limited to the act of writing. The tremulous form may be mistaken for commencing paralysis agitans, but, at the onset, the tremor is confined to writing, and is always conjoined with distinct spasm produced by the act.

Writers' cramp is a disease that is readily imagined. Most persons who have to write much, experience at times some discomfort in the hand after writing, and, since such discomfort is one of the early symptoms of writers' cramp, they are apt to fancy that they are the subjects of the disease, and a slight tendency to spasm is readily imagined in such a case. The most important diagnostic indication is the occurrence of actual visible spasm, since this is seldom absent, even in the early stage. In the cases in which sensory symptoms are ultimately most prominent, they seldom exist alone. If they are isolated, the diagnosis rests on the initial relation to one action and the unusual seat of the pain. In most cases of the fancied disease, when the patients are reassured, the discomfort quickly ceases to be noticed.

PROGNOSIS.—The disease, when well developed, is one in which the prognosis is always uncertain, and often unfavorable. When the affection occurs in a person whose daily bread depends on the act of writing, the early warnings are neglected, either through ignorance or the stern compulsion of necessity, until the morbid action of the centre has become fixed by nutritional alterations almost as ineradicable as those that render permanent the results of its early education. The prognosis is especially unfavorable if the disease has lasted for some time, and has reached a considerable degree before the patient comes under treatment; if perfect rest from writing cannot be secured; and also if no other cause than the act of writing can be traced. It is better if any extraneous and removable cause co-operates, such as defective general health, anxiety, and especially if local causes, such as injury, have distinctly aided in the development of the morbid state. Even, however, under the most unfavorable conditions, recovery sometimes occurs. The writing shown in Fig. 157 was that of a clerk, aged thirty-four, in whom no cause could be traced but excessive writing. He had averaged ten hours a day for twenty years, and the affection had lasted for twelve months before he came under treatment, and yet, as already mentioned (p. 720), recovery has been perfect. The prognosis in cases in which the sensory trouble predominates is good, if the patient can take a long and perfect rest, but unless this can be secured, the neuralgic pain, at first produced only by the act of writing, is likely to extend and become spontaneous. The probability that the other hand will be affected is greater in these cases than in those of purely motor spasm.

Treatment.—Writers' cramp is a troublesome and difficult disease to treat, and the question of its prevention is therefore of corresponding importance. The disease might probably be prevented to a very large extent, if not entirely. We have seen that it occurs almost exclusively among those who write in a cramped manner, in the first or second of the styles mentioned on p. 713, scarcely ever, and only in slight degree, in those who write in the third, and never in those who write in the fourth. If all persons wrote from the shoulder, writers' cramp would practically cease. But the mode of writing is a matter of education, and prevention of the disease rests therefore not with doctors but with teachers. It is probably impossible to teach writing in the first instance in a free manner, but before a boy is allowed to pass from school, he should be got out of the cramped, and taught the free method,—taught, that is, to write from the shoulder. It is also desirable that those who experience any difficulty or discomfort in writing should at once change their style for the freer mode. To make a person realise the characteristics of the free method, I have found the following expedient useful. Let him first draw a line across a sheet of paper; for this, the arm must be moved as a whole from the shoulder. Then let him make the line wavy, next increase the wavy character, and then slightly slope the waves, so as at last to make the line a series of *m*'s—*mmmmmm*. These are letters, and the transition to other letters will then be easy. As I have already said, the object to be aimed at is to write in such a manner that it shall be easy to form an entire line of words without once lifting the pen from the paper. The hand should grasp the pen lightly, and move as a whole. The comfort and ease experienced when this method is acquired are very remarkable.

For the affection itself, treatment, to be effective, should be early. The commencing symptoms often pass away with a brief rest; a month's abstinence from writing at the onset will do more than a year's rest if the disease has continued for six months. When writing is resumed in such a case, it is essential that its style should be changed as just described. The amount of writing should, if possible, also be reduced. It is desirable that a soft quill should be substituted for a steel pen, and that a holder should be employed that is of good size and not smooth on the surface.* Slight symptoms of cramp often pass away at once when this change is made.

In cases in which the disease has become established, rest is still incomparably the most important element in treatment. If writing is continued, every method of treatment fails even to check the progress of the disease. The various mechanical devices which, for a time, aid handwriting are not to be recommended, since they only stave off the impending disability, while the malady is really getting worse, and they thus increase the difficulty of treatment. If, however, prolonged and complete rest from writing is impossible, the patient should learn to write with the left hand. Facility in sinistral writing is acquired with a little perseverance, more readily if the letters are made upright or if the customary inclination is reversed. In this way complete rest may be given to the affected hand. It is true the left hand may, after a time, suffer in the same way, but the tendency to this has been exaggerated by some writers. If it does, the patient is no worse off than if he has not learned to write with the left hand; while if it does not, he may be able to continue to earn his living, and may afford his right hand a fair chance of recovery. The Government clerk mentioned on p. 720 fulfilled the duties of his office for twelve years with his left hand, and although the cramp continued in the right, the left showed no indication of the disease, and at the end of the twelve years he was able to retire on a pension. Besides the case illustrated in Fig. 157, I have met with several others in which practical recovery occurred during the use of the left hand.

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* Quills are too small and too smooth to be held with facility; a quill nib should be used in a holder. The best steel pens are those with turned-up points, as the Waverley. Similar gold pens are made by Mabie, Todd & Co. A good holder is one that is larger than the average size, and is covered with soft india rubber for about two inches, where the pen is held. Large cork holders are also good. The best small holder is one made by Brandauer, of Birmingham, in which the metal part is roughened.

For many persons the use of a type-writer offers the means of abstaining from all the use of the hand that has brought on the disease. The depression of the keys of a good type-writer resembles the movement of the fingers in playing on the piano, but is less fatiguing, and most sufferers from writers' cramp are able to use such an instrument, even with the affected hand, without any unpleasant symptoms, while the facility with which some instruments can be worked with the left hand enables the right hand, if necessary, to have absolute rest. Unfortunately, a type-writer cannot often be available for the work of the ordinary clerk, who is so frequently a sufferer. For most others it offers a great advantage; the depression of the keys, if not continued to fatiguing excess, constitutes a most useful form of gymnastic exercise. In those who suffer from the neuralgic form, however, the pain is often excited by any movement of the arm, and they may then be unable to use a type-writer.*

When impaired nutrition of the nervous system co-operates as a cause of writers' cramp, nervine tonics, especially small doses of strychnia, increase a tendency to recovery, and in all cases may be given, as having an influence in the right direction. Sedatives internally, as a rule, effect little in cases of pure cramp, although hypodermic injections of atropine (one sixtieth of a grain three times a week) have been strongly recommended by Vance. In the neuralgic form, sedatives internally and locally are certainly of service. Indian hemp by the mouth, morphia beneath the skin, or inunctions of extract of belladonna and glycerine, or aconite ointment, are the most useful. I have also found hypodermic injection of cocaine beneficial in some cases when there has been pain about the wrist or forearm. If tenderness of the nerves exists, counter-irritation by blisters over the tender points often does considerable good.

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* The machines with a separate key for each letter, although expensive, are alone useful. Those are to be preferred which have a separate set of keys for capital letters, rather than those in which the capitals are produced by the use of a controlling key, changing the small letters to the capitals.

Electricity has been extensively used, and strongly recommended, in the treatment of writers' cramp. Faradism is of no value, and even harmful, as a rule, but voltaic electricity occasionally gives some relief to the pain. In the spasmodic form Erb recommends the passage of the voltaic current up and down the cervical spine, and from the spine to the muscles. Poore advises the combination of the latter method with gymnastic movements of the hand, and has observed great improvement in some cases from this treatment.* My own experience has led me to assign a low position to electricity in the treatment of the spasmodic form, although I have given it a thorough trial in many cases. Certainly, if the patient goes on writing, it has not the slightest influence on the disease. With rest from writing, there is, in many cases, a tendency to recovery which has not appeared to me to be expedited by galvanism however applied. Gymnastic exercises are often distinctly useful, regular flexion and extension movements of the fingers and hand, for which, with advantage, a "dumb piano" may be used. They should be as different as possible from the movements that induce the spasm, and should never be continued to fatigue. Rubbing, and the combination of rubbing and kneading that is called massage, often gives considerable relief to discomfort, and sometimes lessens the tendency to spasm. It has been recently made the subject of public attention, and has been said to give remarkable results, but it is probable that these were obtained chiefly in cases of the imaginary disorder. While there is no doubt that the treatment often lessens discomfort, in the cases of undoubted writers' cramp that I have known treated in this manner by "experts," the symptoms have not been appreciably influenced.

Among other means of treatment which have been recommended is tenotomy (Stromeyer). In one case (Truppert) no less than fifty tendons were successively divided, and it is said that the patient at the end of the treatment was slightly improved! I have found no good result from rendering the arm for a time immobile by a plaster-of-Paris bandage.

OTHER OCCUPATION NEUROSES.

The general considerations regarding the pathology, causation, and treatment of writers' cramp are applicable, *mutatis mutandis*, to all the other forms of occupation neurosis. In each, pain and spasm may be variously combined, and are at first related solely to the special act.

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* 'Handbook of Electricity.'

Pianoforte players' Cramp occurs chiefly among professionals, and more frequently among women than among men. Both the spasmodic and neuralgic varieties are met with. In the former at first a finger or the thumb does not move with the necessary precision, and may remain extended for a few seconds instead of striking the required note. In the neuralgic form, pain comes on after playing for a short time, and compels the sufferer to desist. Sometimes the pain extends up the arm. Erb has several times met with pain between the scapula and the spine. I have seen one case in which the pain was symmetrical in the outer border of each hand, sometimes extending down the little finger, but never accompanied by cramp, although the patient was liable to the nocturnal cramp in the hands described at p. 704.

Violin players are liable to a similar affection in either hand, especially in that which fingers the strings, less commonly in that which moves the bow, but occasionally in both.

Sempstresses' Cramp.—The act of sewing frequently leads to a similar affection in sempstresses and tailors, usually characterised by rigidity and cramped flexion of the fingers of the right hand, often greatest in the thimble finger. Tingling in the fingers may accompany it, and may cease as soon as the attempt to sew is relinquished. I have met with the same trouble in a sailmaker. In this occupation the needle is held between the finger and thumb, and is pressed through the canvas by a metal plate fixed on the ball of the thumb. The spasm in the fingers caused the needle to slip off the plate, and the attempt to work occasioned pain in the fingers, and in the metacarpo-phalangeal joints.

Telegraphists' cramp (first described by Onimus) affects exclusively those who use the Morse machine, which is worked by pressing down a stop with the finger, so as to make a dot or a dash according to the duration of the pressure. The pressure has to be repeated for each sign, and telegraphists have often to send 50,000 separate signs a day. Spasm is set up, which causes the pressure to be too long maintained, and hence dots become transformed to dashes. One patient managed to go on for two years by substituting his thumb for the finger, and then this became unmanageable; afterwards he used the middle finger and then the closed fist, but the spasm followed, and he had to give up his occupation. The neuralgic variety is sometimes met with, and I have seen one case in which the pain, for six months excited only by the special work, subsequently occurred on other movements. After six months' rest the patient resumed work as a writing clerk, and wrote in comfort for two months. Then the pain returned in connection with writing, rapidly increased, and became spontaneous.

Smiths are also, although rarely, liable to cramp. In one case, an artisan who was accustomed to work metal with a hammer and chisel, found that his left hand, grasping the chisel, gradually passed into a state of tremulous cramp, and became supinated in spite of his effort to prevent it. The spasm was worst when the elbow was flexed, and for a time he was able to work with the elbow extended, but at last he had to relinquish his occupation. In another case, a metal-chaser, who worked with a four-pound hammer, found that, after using it for about an hour, clonic spasm came on in the biceps, and interfered with his work. The use of the hammer in other kinds of work has also induced the affection.

Among other occupations which have been known to lead to the development of cramp, and have suggested names for special varieties, are those of painters, harpists, artificial flower makers, turners, watchmakers, knitters, engravers (in using the burin), masons (in using the trowel), sailors (from pulling on ropes), treadlers, compositors, enamellers, cigarette makers, shoemakers, milkers, money counters, letter sorters, and players on various musical instruments, besides those above mentioned, including drummers, in whom either the extensors of the thumb or the long flexor may be affected (Bruns).

In rare cases two actions are simultaneously and equally affected,—writing and some other. The most common combination is writing and piano playing in ardent amateur musicians; occasionally writing and sewing are both equally deranged. I have met with one obstinate case with chiefly sensory symptoms, in which they were induced by any prolonged use of the hands.

EPILEPSY.

The term "epilepsy" is applied to a disease in which there are convulsions of a certain type, or sudden loss or impairment of consciousness, but in which the convulsions are not directly due to active organic brain disease, to reflex irritation, or to abnormal blood-states, and in which isolated loss of consciousness is not the result of primary failure of the heart's action. In most cases, the change in the brain that causes the convulsions is not visible to the naked eye, and hence the condition is often termed "idiopathic epilepsy." There are cases, however, in which similar convulsions occur as a result of brain disease, such as a spot of old cortical softening, stationary for years and not irritant, but effective by the damage to adjacent nerve-cells; and also of disease, sometimes of long duration, which causes a continuous irritation, such as a depressed spiculum of bone. These cases, when due to a stationary lesion that does not irritate, may resemble epilepsy in their course, and sometimes in their symptoms; they often succeed a distinct attack of hemiplegia, and then are sometimes termed "post-hemiplegic epilepsy."

The convulsions in ordinary epilepsy may consist of tonic or of clonic spasm, or, commonly, of both, but the attitude or movement which results always differs from that which occurs in voluntary movement. On the other hand, in the convulsions of hysteria, the muscular spasm is so arranged as to resemble that which may result from a voluntary action.

Etiology.—After one attack of epilepsy, others usually occur without any immediate discoverable cause. Each fit apparently leaves a change in the nerve-centres, facilitating the occurrence of other fits. The immediate or exciting cause of the disease must be sought, therefore, in the cause of the first fit. But when such a cause can be discovered, it is usually manifestly inadequate alone to produce a convulsion, and can only be effective in consequence of a powerful predisposition. The remote influences to which the disease is due are, therefore, its chief causes. The excitant is no more than the spark which causes a conflagration, rendered possible only by the chemical constitution of the materials ignited.

Remote Causes.—Females suffer from epilepsy rather more frequently than males, in the proportion of about six to five.* An inherited tendency (indicated by the presence in ancestors or collateral relations of epilepsy itself, or of insanity) is traceable in rather more than a third of the cases (35 per cent.), and rather less frequently in males than in females (33 per cent. males, 37 per cent. females). There is a family history of epilepsy in two thirds of the inherited cases; of insanity in one third; of both in a tenth. When there is an inherited tendency, the females of a family are rather more likely to suffer than the males. This is due to two circumstances; the inheritance is rather more frequently from the mother's side than from the father's, and more females suffer when the heredity is maternal, more males when it is paternal. The inheritance is less frequently from the mother personally than from the father, but this is due to the fact that insanity is much less common in the mother than in the father; epilepsy is equally common in the two. Occasionally many members of a family suffer; in one instance no less than fourteen were epileptic, the cases being distributed through four generations. It is very doubtful whether any inherited morbid state, outside the nervous system, predisposes to epilepsy. A proclivity to phthisis has been thought to do so, but the evidence of statistics shows that this is an erroneous inference, due merely to the great frequency of consumption in the community. It is doubtful whether any closer relation exists between epilepsy and the inherited gouty diathesis, to which attention has been lately called. Idiopathic epilepsy, however, sometimes occurs in the subjects of inherited syphilis, without other cause being traceable. A strong predisposition to the disease often exists when there is defective general development of the brain, from any influence acting on it in early life, or as a result of the congenital defect met with in neurotic families and in the offspring of consanguineous marriages.

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* These statements are based on 1450 case [illegible] analysis of which is given in a separate work ('Epilepsy and other Chi [illegible] Diseases,' London, 1881).

Three quarters of the cases of epilepsy begin under twenty years, and nearly half (46 per cent.) between ten and twenty, the maximum being at fourteen, fifteen, and sixteen. One eighth of all cases commence during the first three years of life. After twenty, the annual number gradually falls, but cases commence even in old age (over seventy). In the cases which commence under thirty, females exceed males, in the first ten years by 6 per cent., in the second by 18, and in the third by 12. After thirty the relation is reversed, and the excess of males gradually increases, until after sixty the malady practically begins in males only. It has been said that heredity can be traced only in the cases which commence under twenty years, but this is altogether erroneous. Most cases of hereditary epilepsy begin before twenty, chiefly because most cases of all kinds commence in this period; the proportion borne by the inherited to the non-inherited cases is considerable throughout life. In the first twenty years of life it is nearly 38 per cent., in the second 34 per cent., and in the cases which commence after forty it is 26 per cent. I have known inherited epilepsy to commence at sixty-five and seventy-one.

Immediate Causes.—We must regard, as the exciting cause of epilepsy, that to which the first fit was apparently due; and its influence, as already stated, is no doubt incomparably smaller than that of the predisposition, due to conditions involved in age and heredity. The statements of patients regarding exciting causes have to be received with caution. Excluding the cases in which the relation of the supposed excitant was doubtful, and excluding also the cases originating in infantile "dentition convulsions," some exciting cause can be traced in only 37 per cent. of the cases. In males it is met with more frequently than in females, and the difference between the sexes in this respect is least in childhood, and is greatest in the second decade and in the decline of life. In women between ten and forty, exciting causes are relatively infrequent.

Of the cases that commence in infancy, at least three quarters date from infantile convulsions, ascribed to teething. Such convulsions are known to be the result, in most instances, of the condition of defective development termed rickets. Whether this condition is entirely preventable or not, it would certainly not attain the degree in which it causes convulsions if children were properly fed, and thus a considerable number of cases of epilepsy might, without doubt, be prevented.

Mental emotion—fright, excitement, anxiety—is the most potent exciting cause of epilepsy. The most frequent is fright, which is powerful chiefly in childhood, and at the transition to adult life, while it is very rare after middle life. As might be anticipated, in childhood it is equally effective in each sex, at puberty it is most effective in females, and after twenty it is seldom traceable in men, but is still a relatively frequent cause in women. The profoundly disturbing effect of alarm on the nervous system, deranging, as it does, almost every function, enables us to understand its frequency as an excitant of epilepsy. There is usually an interval between the fright and the first fit. Fright excites hysteroid as well as epileptic fits, and in most of the cases in which a person is immediately "frightened *into* a fit," the attacks are hysteroid. The longer the interval, the larger is the proportion of cases in which the attacks are distinctly epileptic. Other forms of mental excitement occasionally excite the first fit, and most commonly at the period of life, and in the sex, in which fright is chiefly influential; but one emotional cause, prolonged mental anxiety, is effective chiefly in men, and late in life.

Blows and falls on the head, which cause no symptoms of coarse cerebral injury, are sometimes followed by recurring convulsions having all the characters of idiopathic epilepsy. They are apparently due to the general impairment of nutrition in the brain, and lowering of its function, from the mechanical influence on the delicately constituted cells. Such cases are met with at all ages; in childhood equally in the two sexes, but in adult life most frequently in males, doubtless from the risks of occupation. More frequently an injury causes convulsions that begin locally, by causing damage to one spot on the cortex. Epilepsy probably sometimes results from slight bilateral damage to the cortex during birth, insufficient to cause motor paralysis. Exposure to the heat of the sun is frequently assigned as the cause of the first fit, but often merely because the fit occurred in summer sunshine, and without adequate evidence that the attack was thus caused. In some cases, chiefly males, its influence is probable, especially when the fits follow other distinct symptoms of sunstroke.

Acute disease is an occasional cause of idiopathic epilepsy, but the cases ascribed to this influence need careful scrutiny, because "post-hemiplegic epilepsy" often results from a sudden lesion of the brain, during an acute specific disease, probably thrombosis in a surface vein, or embolism consequent on endocarditis (see p. 456). Excluding such cases, epilepsy is a sequel especially to scarlet fever, measles, and typhoid fever in that order of frequency, and more cases are consecutive to scarlet fever (apart from the influence of nephritis) than to all the other acute diseases put together. This, together with the fact that optic neuritis may follow scarlet fever, without any organic change in the brain to cause it, seems to show that the scarlet-fever poison has a special action on the nervous system.

Intestinal worms are a cause of reflex convulsions, and these do not always cease when the worms are expelled. They may continue as idiopathic epilepsy, apparently as a result of a change in the nervous system consequent on the primary convulsions. It is extremely rare to meet with any evidence of a reflex cause in the system of the cerebro-spinal nerves, and in the few recorded cases there has been a manifest and strong predisposition in the brain. A patient with permanent imbecility suffered from fits, beginning in the right hand, after a cut on one of the fingers; pressure on the scar caused pain and excited a fit. The attacks ceased after amputation of the finger.[*] Minor attacks, beginning with a sensation on a scar above one eyebrow,[†] ceased, but only for a time, on excision of the scar. In other cases, arrest has followed the removal of a diseased testicle and of a foreign body in the nose.[‡] In these cases the peripheral irritation seems to have been the exciting cause. Among other rare causes of the first fit are severe digestive derangement, asphyxia, chronic alcoholism, and chronic lead-poisoning. In the two latter the convulsions are primarily due to the toxic influence, but may persist when the latter is lessened or removed by treatment. Analogous convulsions are sometimes met with in chronic renal disease, apart from any acute symptoms of uræmia.

Disturbed or delayed menstruation, at the time of puberty, often coincides with the onset of epilepsy, but it is very difficult to determine the exact relationship between the two. As a rule, when epilepsy is once set up, the establishment of regular menstruation appears to exert no influence on the disease. The fact that so many cases commence at the time of puberty seems due rather to the developmental changes in the nervous system at this epoch than to any direct influence exerted by the organs of generation. No case has come under my notice suggesting that uterine or ovarian disease can be regarded as a cause of epilepsy. Occasionally the first fit occurs during pregnancy, or after parturition, without any secondary cause to which it could be ascribed.

Masturbation in boys is undoubtedly an occasional cause, but the attacks which result from this influence are more frequently hysteroid than epileptic, or they present a hybrid character, so that it is difficult to say in which category they are to be placed.

Syphilis is a frequent cause of chronic convulsions, but these are usually due to organic disease, a meningeal growth or chronic inflammation. Fournier believes that the syphilitic poison, during the secondary period, has a direct action on the nervous system, and may cause pure neuroses, epilepsy among the number. Evidence of this, that will bear a close scrutiny, is scanty. Convulsions may be the only sign of organic changes, which sometimes occur early in the course of syphilis. The occurrence of epilepsy, apparently idiopathic, in the subjects of inherited syphilis, has been already mentioned.

It is not rare for no cause, remote or immediate, to be traceable. This fact will be less surprising when the probable nature of the malady is understood.

Symptoms.—Epileptic attacks are commonly divided into two classes, "major" or severe, and "minor" or slight. These two forms, although clearly distinguished in their general characters, are not separated by any sharp demarcation. In the major attacks (*grand mal*) there is loss of consciousness and severe muscular spasm. In the minor attacks (*petit mal*) there is usually brief loss of consciousness, often without any muscular spasm, sometimes with slight spasm, and very rarely there is slight spasm, or some sudden sensation, without loss of consciousness. In severe attacks the patient, if standing, falls to the ground (hence the old English name, "falling sickness"); in slight attacks he may or may not fall. In very severe fits muscular spasm and loss of consciousness are simultaneous in onset, but in less severe fits the muscular spasm may commence before consciousness is lost; the patient is then aware of the onset. Still more frequently the spasm and loss of consciousness are preceded by some sensation. The sensation, or spasm, which informs the patient of the oncoming attack, constitutes the "warning" or "aura" of the fit.

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[*] Guder, 'Zeitsch. f. Psych.,' 1890.

[†] Rik, 'Zeitsch. f Heilk.,' 1889.

[‡] Hansdale, 'Am. J. Med. Sc.,' 1889.

Major Fits.—At the onset of the severe fit the spasm is tonic in character,—rigid, violent, muscular contraction, fixing the limbs in some strained position. There is usually deviation of the eyes and of the head towards one side, and this may amount to rotation involving the whole body, and may actually cause the patient to turn round, even two or three times. The features are distorted; the colour of the face, unchanged at first, rapidly becomes pale, then flushed, and ultimately livid, as the fixation of the chest by the spasm stops the movements of respiration. The eyes are open or closed; the conjunctiva is insensitive; the pupils dilate widely as cyanosis comes on. As the spasm continues, it commonly changes in its relative intensity in different parts, causing slight alterations in the position of the limbs. When the cyanosis has become intense, the fixed tetanic contractions of the muscles can be felt to be vibratory, and the vibrations gradually increase until there are slight visible remissions. As these become deeper, the muscular contractions become more shock-like in character, and the stage of clonic spasm is reached, in which the limbs, head, face, and trunk are jerked with violence, and through similar spasms in the tongue and muscles of the jaws the former is often bitten. The shocks of spasm effect slight movements of the thorax, so that air is expelled, and saliva is frothed out between the lips. The air that enters the lungs is at first insufficient to lessen the lividity, and the patient may seem to be at the point of death, but as the remissions become deeper, more air enters the chest, and the lividity gradually lessens. In becoming less frequent, the contractions do not become less strong, and the last jerk is often as violent as those which have preceded it. At last the spasm is at an end, and the patient lies senseless and prostrate, sleeps heavily for a time, and then can be roused. Urine frequently, and fæces occasionally, are passed during the fit.

In some cases the spasm is more deliberate in its onset. Instead of commencing simultaneously in all the muscles of the body, it begins in one region, as the face or arm, and thence spreads, first to the limbs on the same side, the head and eyes being turned towards that side, and then, lessening on the side first affected, it invades the limbs on the opposite side, with a corresponding rotation of the head. Such attacks may commence with tonic spasm, or they may commence by, and sometimes consist only of, clonic spasm. This form of convulsion is that which (as we have already seen, p. 90) is most common in organic cerebral disease, such as tumour, but it is also occasionally met with in idiopathic epilepsy. In such cases consciousness is often lost late, so that the patient is aware of the commencing spasm.

Minor Attacks.—The slight attacks of epilepsy may be characterised only by loss of consciousness. The individual suddenly stops in his occupation, looks strange for a moment, and then goes on with what he is doing, may even finish a sentence that he had commenced, and be aware of what has happened only by finding that he has dropped something which was in his hands, or that he is the object of anxious observation. Sometimes there is pallor at the moment of the attack, more frequently after the onset, and very commonly the face subsequently becomes flushed. Very often, moreover, the slight seizure is heralded by some sensory warning or aura, such as precedes the severe attacks. There may also be slight visible spasm, such as putting the arms forward and bending the head down, or there may be slight convulsion in the part in which the spasm commences in the severe attacks, and, in some cases, the minor attack may consist only of such spasm, without loss of consciousness. After a slight attack is over, the patient may be at once quite well. Often he is stupid and dull for a little time, and sometimes proceeds to perform some action in a dream-like, automatic manner, such as undressing himself, retaining afterwards no recollection of what he has done. Occasionally this stage is attended by passion and violence, a brief maniacal condition constituting one form of epileptic mania. After an attack, severe, or more frequently slight, the patient may pass into a state of actual hysteroid convulsion, and in such cases the minor epileptic seizure may be unnoticed, all the obtrusive phenomena of the attack being of a hysteroid character.

Symptoms in Detail.—*Precursory symptoms* occasionally precede a fit for some hours or days, and indicate to the patient or his friends that an attack is impending. The most frequent of these indications are sudden jerks of the body or limbs, persistent giddiness, flashes of light before the eyes, irritability of temper, or an unnatural degree of appetite. In rare cases there is considerable mental change, even with delusions, for some hours before an attack.

Mode of Onset; Warning; Aura.—The word *aura* (vapour) originated with the old Greek theory that the fit began by the ascent of a vapour up the vessels of the limb. In later times it was thought that the sensation felt in the periphery does begin there, as a disturbance ascending the nerves; and the opinion was thought to be confirmed by the fact that a ligature around the limb would arrest the ascending aura. But the fact (observed first in the beginning of this century) that the commencing fit may be equally arrested when its cause is a tumour of the brain, has shown that the aura is merely the effect on consciousness of the commencing discharge in the brain, either a direct effect, felt as a sensation and referred to the periphery, or an indirect effect, due to actual spasm at the periphery. But the fact that the aura is the result of the commencing central process renders its study of great importance, since it gives us information of the functional region of the brain in which the process of the fit begins.

Some form of aura is met with, at least occasionally, in about one half of the cases of epilepsy. In the other half the loss of consciousness always occurs so early that the patient is unaware of the onset of the fit. We may provisionally group the various auræ into certain classes for convenience of description. The classification must be, in the main, empirical.

(1) The commencement of the attack may be by a motion or sensation in some part of one half of the body, most frequently in the arm, less frequently in the face or leg, occasionally in the tongue, very rarely in the side of the trunk. Such local commencement is not frequent in idiopathic epilepsy, although it is sometimes met with; it is the common mode of onset in cases of organic brain disease. In any part, the first symptom may be a spasm, or a sensation, or both. In the upper limb the aura usually commences in the hand. When it is a spasm, it generally commences in the hand as a whole, less commonly in the arm as a whole, rarely in the shoulder. When a sensation, it is usually described as "numbness," "tingling," &c., and commences generally in a definite part of the hand,—thumb, fore-finger, all the fingers, palm, or wrist: never higher up the arm. Sometimes there is a sensation of motion without actual spasm. The aura, whether motor or sensory, if it commences in the hand, may be felt to ascend the arm and may pass to the head, trunk, or leg, before consciousness is lost. Commencement in the leg is less frequent, and presents the same variations in character and seat; the sensation or spasm may pass, before consciousness is lost, up or down the limb (according to the place of commencement), or to the trunk, arm, or head. When the aura passes from the arm to the leg, or *vice versâ*, it may do so by two modes: it may pass from one limb to the other through the trunk, and then pass *down* the limb secondarily affected; or it may commence at the extremity of the second limb, and thus pass *up* both. In the former case, as a rule, a pure sensation leads; the course of the aura is determined by the discharge in the sensory centre, in which no doubt the representation of the cutaneous nerves is as continuous as their distribution in the skin. Sometimes a purely sensory aura (discharge limited to a sensory centre) may pass up one limb, along the side of the trunk, and down the other limb, and only

when it reaches the extremity of this latter is spasm added, which may pass back up the limb, the discharge extending to the related motor centre only when it reaches the limit of the sensory centre.

Fits begin in the face much less frequently by a sensation than by spasm; the latter is generally in the zygomatic muscles, sometimes in the orbicularis. In slight attacks on the right side, the commencement in the face is often associated with inability to speak. An aura in the tongue is usually sensory; it may be associated with movement in the jaw or with nausea, and in these combinations we may trace physiological associations.

(2) Bilateral and general warnings. Fits occasionally commence by a sensation or motion in the limbs on both sides simultaneously. An aura felt in the trunk and not distinctly visceral is rare, and is generally referred to the spine. General tremor or shivering, and general starts or jerks, are occasional warnings; and so are a general sense of malaise, of powerlessness, of heat, and a feeling of faintness.

(3) Visceral and pneumogastric warnings. A frequent form of aura is that which is referred to the internal viscera, and especially to those which are in the region of the pneumogastric nerve, including the respiratory portion of the spinal accessory. The most common is some sensation at the epigastrium, usually vague, but sometimes actual pain. Rarely it seems to commence lower in the abdomen; occasionally it is referred to the left of the middle line, scarcely ever to the right. When the sensation is actual pain, it remains at the epigastrium until consciousness is lost; it is sometimes associated with nausea, and then seems referable to disturbance in the central relations of the gastric division of the vagus. When the sensation is other than pain, it may also only be felt at the epigastrium, but frequently it seems to ascend through the chest to the throat or head. On reaching the throat there is a sensation of choking, apparently identical with the globus hystericus. The warning may also be this sensation of choking without any preceding epigastric aura. Now and then other vague feelings are described in the throat or thorax. These auræ, ascending to or felt in the throat, seem to be the expression of a disturbance of the central processes connected with the respiratory function of the pneumogastric. Another organ within the innervation of the vagus is the heart, and various cardiac sensations, such as palpitation, or pain, or vague discomfort, sometimes constitute the aura.

(4) Cephalic sensations. A sensation of giddiness, vague or definite vertigo, is a very frequent aura, and is often associated with actual turning of the head and eyes, seldom of the whole body, more frequently to the left than to the right. In bilateral fits the convulsion is never quite equal on the two sides, and the deviation of the head is perhaps due to the inequality of discharge, and the vertigo its sensory effect (see "Vertigo"). Giddiness is occasionally associated with nausea, and sometimes with loss of sight. Other sensations referred to the head are also common; actual pain, usually indefinite in seat,

sometimes a sense of "rushing of blood to the head," or vague "heaviness." A sudden sense of somnolence is also sometimes described.

(5) Psychical auræ occasionally herald attacks, and may consist in an emotion or an idea. The emotion is uniform in the same case, some form or degree of fear. It bears no relation to emotion as a cause of the first fit. The idea is usually too vague or imperfectly remembered to be described in words. Occasionally the psychical aura consists of a vague "dreamy state;" sometimes it is a sudden sense of strangeness—familiar objects seem unfamiliar. Very rarely, there is a sudden sense that whatever is happening is morally wrong.

(6) Special sense auræ are very frequent and important. Olfactory sensations are rare; when experienced they are usually unpleasant in character, a "sulphurous," "fœtid," or "indescribable" smell. When due to organic brain disease, this has sometimes involved the uncinate gyrus. Gustatory auræ—a sour, bitter, or metallic taste—are still more rare. Flavours (an olfactory sensation) are never combined with a sensation of an odour as a warning, but sometimes are with a true gustatory sensation—a fact of much physiological interest. An auditory warning is more common. There may be a sudden loss of hearing—a strange stillness, for a moment, before consciousness is lost. More frequently there is a warning sound, usually of low elaboration—a crash, whiz, a hiss or whistle, and the sound may seem to get louder and nearer until the patient knows nothing more. Very rarely a more elaborate sensation is described, or music, or words, and the sensation is usually the same before each fit. One patient described, as the constant aura, a curious perception of hearing words without any sensation of hearing them.

A visual warning is twice as frequent as all the other special sense auræ together. It may be sudden loss of sight; but more frequently it is a visual sensation, a flash of light, or sparks, or flashes of colour. Usually many colours are seen, sometimes one only. Red and blue are the most frequent; no other colour is seen alone, and both are never absent. Occasionally the visual sensation is much more elaborate, a vague vision of some beautiful place, or a definite image of some object, an old woman with a dress of a certain colour, ugly faces, animals, &c. Objects actually before the patient may appear to approach, or more frequently to recede, as the fit is coming on. The recession is apparently due to a slight degree of the inhibition of the visual centre, which, in greater degree, causes the loss of sight. It is noteworthy that lights may be seen after sight is lost, an instance of "discharge" in an inhibited centre, and an important fact in connection with the physiological pathology of the disease. Various special sense auræ may be combined.*

Very rarely an attack is preceded by some co-ordinated movement. A patient, for instance, may invariably commence running, and after running a few yards, fall down in a fit,—the "epilepsia cursiva" of Boötius. I have known a patient always to turn round and retrace his steps immediately before an attack, and another would spring up and jump about the room for a few seconds. In one instance the forward run before the fall was preceded by senseless laughter and irregular movement of the arms.*

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* Many illustrations of the different kinds of warning will be found in my treatise on "Epilepsy."

A peculiar sound, the "epileptic cry," is produced at the onset of the attack in some cases. It may be a harsh weird scream, but more often it is a sort of prolonged laryngeal groan, not very loud, but very characteristic. The sounds are probably due to narrowing of the glottis at the moment when air is expelled from the chest by the tonic spasm. As a rule the patient is not aware of the cry, but sometimes he can hear and remember it, although he cannot prevent it.

The convulsion, as a rule, begins by tonic spasm, which causes rotation of the head and deviation of the eyes and of the mouth to the side on which the convulsion is most intense. The posture of the limbs varies. Commonly the arms are slightly abducted at the shoulder, the elbow and wrist are flexed, and the fingers are flexed at the metacarpo-phalangeal joints, extended at the others, the thumb being adducted into the palm, or pressed against the fingers. The position is thus nearly that seen in tetany. The legs may be extended, but often there is slight flexion at the hip- and knee-joints. Sometimes initial extension of the legs gives place to strong flexion in the later stages of the fit. Usually the limbs of the two sides do not perfectly correspond in position, but the difference is slight. In other cases the arms are extended, and in some the fingers are flexed at all joints, the fists being "clenched." In others again, instead of a combination of flexion and extension, flexion predominates throughout. The head is bent forwards, the arms and legs are strongly flexed, so that the fists are in contact with the chest, and the knees with the abdomen. At the commencement of such flexor fits the patient often falls forwards. In rare cases the arms are raised up above the head at the onset of the attack, and may be kept in that position throughout, or put straight forwards. The neck, in these fits, is often bent backwards; the legs may be extended, or one or both may be flexed. The violence of the tonic spasm is often very great; the shoulder may be dislocated by it, and when this accident has once happened it is very apt to recur. It is generally during the stage of clonic spasm that the tongue is bitten; it is pushed by the spasm between the teeth, while the jaws are jerked by the contraction in the muscles of mastication. Very rarely it is bitten during the tonic stage. Now and then, instead of this shock-like clonic spasm, into which the tonic spasm gradually passes, the clonic spasm is finer in range and quicker in time, and is superadded to, instead of superseding, the tonic spasm. The latter continues while the strained rigid limbs are agitated by the coarse tremor. This form of clonic spasm is most common in the cases which present extension in the arms and flexion of all the joints of the fingers.

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* Marrey, 'Rev. de Méd.,' 1889.

In some epileptic fits there is but one form of spasm, either tonic or clonic. As a rule, those which consist only of tonic spasm are general fits of slight severity. A patient falls unconscious, is rigid for a few moments, and then is better. Severer tonic fits are sometimes seen—the "tetanoid epilepsy" of Pritchard.

The attacks which consist only of clonic spasm are slight fits of partial distribution, beginning in, and often confined to, one limb, especially the hand and arm. They are less common in cases having the character of idiopathic epilepsy than in organic brain disease. Severer fits consisting only of clonic spasm are very rare. Rarely fine general tremor constitutes all the convulsive phenomena, or succeeds tonic spasm (Féré).

The course of the spasm in fits which begin locally has been alluded to in the account of the modes of onset. The relation of the spasm to the muscles of unilateral and bilateral use is an important point, which has been already considered (p. 90).

The slight convulsion at the onset of fits which begin deliberately seems to cause little pain, but occasionally the pain felt in such cases is very great. In some of these cases it is probable that the pain is due to discharge in the sensory centres.

Other Symptoms of Attacks.—The pupils are said to be occasionally contracted at the onset of a fit, but such contraction is certainly not invariable. As a rule, as soon as the tonic stage is well established, the pupils are dilated, and continue so until the fit is over; the iris does not contract to light. When signs of consciousness can be elicited, the dilatation ceases, and it is not uncommon after the fit for the pupil to present alternate contraction and dilatation every one or two seconds, continuing for a few minutes, a phenomenon first pointed out by Reynolds.

The passage of urine during an attack, already mentioned, is not due merely to the state of the bladder or rectum, or to the loss of consciousness, but is the result of some peculiarity in the convulsion, since it occurs invariably in some patients, never in others. The pulse may be feeble at the onset, but I have never noted an initial failing, although I have several times had my finger on the pulse when a fit came on. A tracing published by Voisin shows that the heart's action may be perfectly normal during the stage of the aura. A stoppage of the pulse has been described by Moxon, but is certainly altogether exceptional. As the muscular spasm becomes considerable the pulse is increased in frequency and in force, but falls when the fit is over.* The face is often pale at the onset, but pallor is less constant than is currently asserted. It may come on after, and not before the commencement of the tonic spasm, but as this continues the face becomes congested and then cyanotic. The bloated dusky aspect of the face, with features distorted by the spasm, renders the appearance of the patient most alarming to those unaccustomed to the disease. When the clonic spasm becomes completely intermittent, air is changed in the lungs and the cyanosis lessens. During the course of the fit, the surface often becomes covered with sweat. It is doubtful whether there is any change in the retinal vessels until the veins become swollen in the course of the venous congestion. In convulsions which begin locally, I have watched the fundus and have seen that there is no initial alteration in the state of the vessels.

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* In a case in which Dr. Money was listening to the heart at the time of the onset, its sounds were regular and vigorous up to the time when pallor and tonic spasm came on, when he had to leave off listening. The pulse continued full and steady throughout, but was rather infrequent. (Oral communication.)

Excitants of Attacks.—In some patients attacks may be excited by certain influences, but such cases are as rare in epilepsy as they are common in hysteria. The excitants which have come under my notice are emotion, a "startling" noise, a bright light, and voluntary motion after rest. It is excessively rare for cutaneous irritation to excite a fit. Epileptogenic zones, analogous to those of Brown-Séquard's guinea-pigs, are practically unknown.[*]

Arrest of Fits.—Attacks which begin deliberately can sometimes be cut short, and the means by which this may be effected are of considerable interest, and of some practical importance. Those which commence by a bilateral, visceral, cephalic, or special sense aura can rarely be arrested; but now and then they can be stopped by some muscular exertion, as by walking quickly, or by some strong sensory impression, such as the application of ammonia to the nostrils, swallowing a mouthful of common salt, or by the inhalation of nitrite of amyl. The attacks that can most frequently be arrested are those which commence by a motion or sensation in the hand or foot. The oldest and usual method of effecting this is by a ligature around the limb above the seat of the spasm or sensation. The strong peripheral impression apparently raises the resistance in the nerve-cells of the affected part of the brain, and so arrests the spread of the discharge. The influence must be exerted first on the sensory centre, and through this on the motor centre. If the commencing fit has got beyond the part to which the ligature has been applied, the attack is not arrested. A ligature acts better than other peripheral impressions, probably because it is most readily applied, and because the cutaneous nerves are stimulated in the entire circumference of the limb, and so influence the entire extent of grey matter in which the discharge is advancing. Occasionally, a more limited cutaneous impression, a pinch or prick, has the same effect. The fits which begin by spasm may sometimes be arrested in another way—by preventing the movement, and forcibly extending the contracting muscles, sometimes by rubbing them. The mechanism is probably the same. Rubbing has a tendency to lessen all forms of spasm, even that of tetanus. Both methods of arrest were known to the ancients.

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[*] See, however, a curious case recorded by Hughlings Jackson ('Proc. Med. Soc. Lond.,' vol. x, 1887. p. 78).

Conditions after Attacks.—The coma, into which the patient usually passes at the end of an epileptic fit, often continues as heavy sleep, lasting for a quarter of an hour or longer. After the first few minutes the patient can be roused. The sleep is often followed by a severe headache, enduring for several hours or the remainder of the day. Occasionally the sleep is not followed by headache, but if the patient is roused and is not allowed to sleep, the pain in the head is severe. In some cases there is neither sleep nor headache.

For a few seconds after a severe fit, reflex action in the limbs remains absent. Tickling the sole no longer causes a movement of the leg, and the loss may involve the muscle-reflex action, so that the knee-jerk can no longer be obtained, as Westphal first showed. But this stage of loss is succeeded by temporary increase in the myotatic irritability, so that the knee-jerk is excessive and the foot-clonus may be obtained. In most cases this increase is alone to be detected. It may be present only on the side on which the convulsion was most severe, *i. e.* on the side towards which the head is turned (Beevor). It lasts a few minutes and then passes away. These phenomena are probably the result of temporary exhaustion of the centres concerned —of the muscle-reflex centre in the case of loss, of the controlling structures (the terminations of the pyramidal fibres)* in the case of excess.

Loss of motor power, paralysis, may succeed a fit of epileptic type —post-convulsive paralysis, as already described (p. 92). It is most distinct after unilateral convulsions (and constitutes the "epileptic hemiplegia" of Todd), but the general prostration after a bilateral convulsion is probably analogous. After a severe fit it may be due to exhaustion of the nerve-elements, but the transient palsy that succeeds a very slight fit must be ascribed to inhibition of the motor centres. In many of these cases there is conspicuous sensory discharge, "tingling," &c., passing, for instance, up the arm, and down the side to the leg; there may, indeed, be no motor spasm. In such a case of purely sensory discharge, the arm may be for a time almost powerless. Just as such a discharge in the sensory centre may, as we have seen, set up secondary discharge in the motor centre, so it may, when slight, merely inhibit the centre. Loss of speech sometimes occurs after right-sided seizures, and probably has the same inhibitory origin. These forms of transient palsy must, however, be distinguished from palsy due to cerebral hæmorrhage caused by the fit (an excessively rare event), and also from the condition in which convulsions merely attend the onset of an acute cerebral lesion.

Automatic action and hysteroid phenomena are very common after slight attacks of epilepsy, and occasionally succeed a severe fit. For instance, a girl aged seventeen had suffered from six years of age from severe epileptic seizures with tongue-biting; at the age of puberty the attacks, without change in character, were succeeded by hysteroid convulsions. In one that was seen, she suddenly fell with a scream; her head was turned to the left, the limbs were rigid, the face cyanotic, clonic spasm succeeded, with frothing at the mouth; the tongue was bitten. The fit lasted two minutes and a half. She then lay unconscious, breathing heavily, and still frothing at the mouth. Then she opened her eyes, looked strange, kicked and threw her arms about, dashed her head, and arched her back for four minutes. She then went to sleep, and slept for an hour.

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* See vol. i, p. 201.

The temperature may be raised half a degree or a degree by a severe convulsion, or it may present no alteration. When attacks follow each other at intervals of only a few minutes (status epilepticus), the temperature may rise to 105° or 107° (Bourneville). The strain may cause small vessels to rupture, usually in the face and conjunctiva, so that the face may be spattered, as it were, with small hæmorrhagic points. After death in a convulsion, similar extravasations may be found in internal organs.

Vomiting occurs after fits, in some patients. It is a dangerous symptom, on account of the liability of food to get into the larynx in the state of insensibility. Excessive hunger is an occasional symptom after an attack, and a patient who has had a hearty meal before a fit will eat another directly after it. The urine is rarely altered in its constitution by an epileptic seizure. Now and then a trace of albumen or of sugar may be found, but the frequency of this has been greatly exaggerated; each is rare. There is no necessary increase in the amount of urea. It has been said that every fit causes a loss of body-weight (Kowalewsky), but this has not been found by others (Lehmann, Beevor, &c.).

Minor Attacks.—The slight attacks of epilepsy vary much in character, and hence patients speak of them under various designations, such as "sensations," "faints," "losses," "turns," "giddiness." Hence, also, their nature is often not recognised by the patients or their friends. As a rule there is brief loss of consciousness, and this, as already stated, may constitute the only apparent symptom. Often, however, there is a slight stoop forwards, or a slight quivering of the eyelids. The patient may or may not fall. The colour of the face, in many cases, undergoes no change; there is scarcely ever pallor at the moment of onset, but in a second or two the face becomes slightly pale in many cases, and when the attack is over, whether there has or has not been pallor, the face commonly becomes a little flushed. Nor is there any change in the retinal vessels. In one case I was observing the vessels when an attack came on; the eyes were open and fixed, and for several seconds after the onset, as well as before it, I could not perceive the slighest change in their size. In many patients the loss of consciousness is preceded by some warning sensation, and it is from this circumstance that the minor seizures are so often termed "sensations." The patient may know nothing of the loss of consciousness, and be aware only of the sensation. Very often a warning occurs only before the minor seizure, the severe fits coming on so rapidly that loss of consciousness is the earliest symptom. When each kind of attack is preceded by an aura, this is usually similar in character, although, as already mentioned, it may be more elaborate before the slight than before the severe seizures. It is rare for the aura of each to be altogether different.

The following list comprehends the chief characteristic features of minor seizures. They are arranged in the order of frequency. In most cases there is, in addition, brief loss of consciousness. The first of the list occurs in one third of the cases, and the second in a sixth, so that these two are the characteristics of about half the minor epileptic seizures met with.

1. Sudden momentary unconsciousness, or "fainting" or "sleepiness," without warning.
2. Giddiness.
3. Jerks or starts of the limbs, trunk, or head.
4. Visual sensation or loss.
5. Mental state; sudden sense of fear, &c.
6. Unilateral peripheral sensation or spasm.
7. Epigastric sensation.
8. Sudden tremor.
9. Sensations in both hands.
10. Pain or other sensation in the head.
11. Choking sensation in the throat.
12. Sudden scream.
13. Olfactory sensation.
14. Cardiac sensation.
15. Sensation in nose or eyeball.
16. Sudden dyspnœa.
17. General "indescribable" sensations.

While giddiness is thus one of the most common sensations, it is only present in a small proportion of the total number, and it is a mistake to employ the term "epileptic vertigo" (as it has been employed) as a general designation for attacks of minor epilepsy. The sense of vertigo usually involves an apparent movement, sometimes in the patient, sometimes in objects seen; the direction of these apparent movements is usually the same;* the vertigo is sometimes attended with actual rotation. Special-sense auræ are also frequent in minor epilepsy, and present the characters already described; the visual sensation is the only frequent one. The epigastric sensation is much less common than before severe fits.

Loss of consciousness is the rule, but exceptions are often met with, in which there is merely obscuration of consciousness for a few seconds, and no absolute loss. In the cases in which the minor attacks consist of sudden starts, or of a visual sensation, consciousness may be apparently unaffected.

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* The explanation of the fact will be given in the account of vertigo. It may be well to remind the observer that the term "giddiness" or "dizziness" is often applied to any obscuration of consciousness, and the exact character of the sensation should always be ascertained.

Urine is often passed during a minor attack; in such cases there is almost always loss of consciousness, but one patient was always aware of it, although unable to prevent it. In some cases micturition is almost invariable, in others it never occurs. The accident is far more common in females than in males; perhaps because the process can occur more quickly on account of the shortness of the urethra.

In some cases the minor attacks may be accompanied with slight convulsive spasm, tonic when general, but usually clonic when it is local. In such cases attacks occur which present every gradation between the slight and severe seizures. The degree which is the maximum in one case may be the minimum in another.

Conditions after Minor Attacks.—The return of consciousness to its average degree often occurs slowly; the patient is dazed and stupid for a few minutes, makes random remarks, and occasionally performs, in an automatic manner, some action of which he afterwards retains no recollection. Such automatic action was formerly regarded as constituting the minor attack of epilepsy, and these cases were therefore called "masked epilepsy" (Esquirol), or "epilepsia larvata" (Morel). I believe that the old view is not altogether untrue; that in some cases imperfect loss of consciousness, with automatic action, does constitute the minor seizure, without any initial stage more distinctly epileptic. But it is certain that, much more frequently, the automatism succeeds a slight attack, and is really a post-epileptic phenomenon. It has been ascribed to the temporary failure of the highest centres to control those next below them, which consequently act in an automatic manner (Anstie, Thompson-Dickson, Hughlings Jackson). The condition is not merely of clinical interest, but also of medico-legal importance, since the performances may be complex, and may have all the aspect of deliberate volition; the initial epileptic seizure may be unnoticed by those around, and even unknown to the patient. One of the most common actions is that of undressing, which is occasionally very inconvenient. It is possibly suggested by a feeling of illness, and so likewise may be another occasional action, an attempt to walk upstairs, which is also awkward if (as happened in two cases) the shelves of a dresser, or the dinner-table, is mistaken for the staircase. A very common action, still more equivocal in its aspect, is to put in the pocket any object which may be near, irrespective of its ownership. This gave rise at first to grave suspicion in the case of one of my patients, a draper's assistant, who had gone to a new situation. Very complex actions may be performed in this state. I have known a carman, after an attack, to drive through the most crowded parts of London without any object, but also without any accident. Occasionally the automatic action displays emotion, and even anger and violence. One patient struck a friend who was with him a violent blow on the face, and was in consequence taken to the police station. Another, a woman, immediately after a fit, threw her baby downstairs. Without doubt many crimes have been committed in this state, and the point has formed a medico-legal question at many criminal trials.

Instead of presenting such automatic action, some patients pass, as already mentioned, into a state of violent hysteroid convulsion. This sequel occurs chiefly at the age at which hysteria is met with, under thirty-five. It is most common in young women, frequent in boys and girls, occasional in young men. Hence it is evidently the result, not merely of the preceding epileptic fit, but also of the presence of the cerebral state which underlies other manifestations of hysteria. Some patients have similar attacks at other times as well as after their epileptic fits. In others there are no separate hysterical phenomena, the hysterical tendency being apparently insufficient to lead to independent symptoms, although it manifests itself during the morbid state immediately after an epileptic fit. As an example of these phenomena may be mentioned the case of a girl, aged fourteen, who had had many severe epileptic fits accompanied by tongue-biting, and also slighter attacks followed by hysteroid convulsions. I witnessed one of the latter. When speaking to me she suddenly stopped, bent forwards, and remained still for a few seconds; then she suddenly threw her arms about, stamped with her feet, and became stiff and rigid for a few minutes in a characteristic hysteroid convulsion. It is often said that attacks in childhood were slight, and at the time of puberty became severe, when further inquiry shows that the severe fits consisted only in the addition of hysteroid convulsion to the slight attacks. There is another post-epileptic action which is automatic, and may occur alone or with hysteroid convulsion, and is important on account of its danger—a tendency to turn over on the face. If the patient is in bed, suffocation may easily occur during the comatose sleep that follows, and without doubt many epileptics have died from this cause. Some patients never present this tendency, others always do, and their friends should be warned of its danger. Minor attacks, when frequent, may entail great physical weakness, so that the patient can scarcely walk. In other cases no such effect is produced.*

Mental Disturbance in Epileptics.—The occurrence of transient "epileptic mania" as a sequel to attacks has been already mentioned. It is uncertain whether such an outbreak may take the place of a fit, but we are not at present justified in denying its possibility. In this condition the patient is often violent and homicidal, and, although perfectly sane in the intervals, is one of the most dangerous of lunatics. The mania is usually brief, often lasting only a few minutes, rarely more than an hour. But occasionally, after a fit or a series of fits, mental disturbance may come on and last for several days, a state of dementia, or of mania, with delusions or hallucinations, often with irritability and violence. It may even occur during the temporary cessation of attacks.

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* It has been said that there is commonly to be found, in epileptics, some restriction of the fields of vision, especially after a fit, and that it is greatest in the upper inner half of the left eye and the lower outer half of the right eye (Otolonghi, 'Lombroso's Archiv,' 1890). I have met with general reduction of the fields to a third, after an attack.

The interparoxysmal mental state of epileptics often presents grave deterioration, and this is one of the most serious and most dreaded effects of the disease. In its slighter degree there is merely defective memory, especially for recent acquisitions. In greater degree the intellect suffers generally, and there is often defective moral control. Mischievous restlessness and irritability in childhood may develop to vicious and even criminal tendencies in adult life. Every grade of defect may be met with, to actual imbecility. The mental state is not, in all cases, entirely the result of the attacks of epilepsy. In some it is, in part at least, the expression of a cerebral imperfection, of which the epilepsy is another manifestation. In such instances mental defect may exist before the occurrence of the first fit, or may follow attacks very rapidly. In other cases the failure is apparently produced solely by the attacks. It succeeds them in time, and may lessen when treatment renders them less frequent.

A comparison of cases with considerable mental defect, and others in which no mental failure was noted, shows that the deterioration cannot be ascribed to any one element in the disease. The most influential agents are the early commencement of the disease, its duration, and the frequency of attacks. The tendency is greater in the cases that commence in childhood than in those which commence between ten and twenty, and greater in those which begin in the first than in the second five years of life. The influence of duration is shown by the fact that, of the cases with mental failure, nearly three quarters, and of those without, less than half, had lasted more than four years. The influence of frequency of attack is shown by the fact that the average interval in the weak-minded cases was fifteen days, in the others twenty-six days. On the other hand, the influence of form of attack (major or minor), of heredity, and of sex is not great. Considerable failure is often seen in patients who have only minor attacks, but extended observation does not show that it is more common in such cases than in others. That there is no necessary relation between any of these conditions and mental failure is shown by the fact that each one may be met with in high degree without any impairment of the intellect. On the other hand, there is no condition in which mental deterioration does not occasionally occur. The conclusion is that mental failure is determined less by single conditions than by their combinations, and it is probable that a more potent cause than the attacks themselves is a predisposition to suffer under their influence, a predisposition that is related to the ultimate causes of the disease, rather than to its developed characters.

When attacks that have occurred during many years, stop suddenly, whether the stoppage is spontaneous or due to the influence of drugs, patients may become dull, forgetful, sometimes irritable, and sometimes half idiotic. The effect is often ascribed to the remedy used, especially if this is bromide; but it seems to be due rather to the general depression of cerebral function by the condition of the nerve-cells, which, by repeated discharge, have developed a tendency to over-production of nerve-force, and this is no longer released in the fits. A fit may completely remove the state. It is common for patients to say that they feel better when they are having fits than when they are not. The cerebral depression is probably increased, when it is not caused, by full doses of bromide.

The general health of epileptics may be perfect. Often there is some defect, especially in the functions of the digestive organs. The circulation is sometimes feeble, the pulse small, unduly frequent, and very often slightly irregular. No abnormal state of the pupil or retinal circulation can be found with any special frequency in the intervals between the attacks.

Post-hemiplegic Epilepsy.—In a considerable proportion of the cases of epilepsy the convulsions succeed an attack of hemiplegia, sudden in onset, and therefore presumably due to a vascular lesion. The palsy may lessen or disappear, and the convulsions, continuing years after, may resemble those of the idiopathic form, and are often supposed to be of this nature. Hence it is convenient to consider them here.

Recurring convulsions may follow hemiplegia at any age, but are far more frequent after that which occurs in infancy than after hemiplegia in adult life. In two thirds of the cases the onset is before five years of age, and in nearly half it is during the first two years of life. In the cases that date from infancy, females are twice as numerous as males; after five years of age, there is little difference in the frequency with which each sex suffers. The paralysis, in the infantile cases, is more frequently on the left side than on the right, but after the fifth year it is as frequently on one side as on the other. The conditions of onset, and the probable nature of the lesion, in the infantile cases, have been already considered (p. 456). In later life, when there is any indication of the cause of the hemiplegia, this also (heart disease, rheumatic fever, constitutional syphilis, the puerperal state) points to vascular obstruction. In softening from this cause, the brain tissue adjacent is usually damaged by the collateral congestion, and it is easy to understand that the nerve-cells may suffer a permanent change in their nutrition and function, causing instability, perpetuated by their repeated discharge. Moreover, acute softening often affects the cortex of the brain, where organic changes most frequently cause convulsions. In a few cases the condition is said to date from birth; in most of these the labour was difficult, and then meningeal hæmorrhage, with laceration of the cortex, is probable (see p. 413).

In half the cases, convulsions, often repeated and severe, attend the onset of the hemiplegia. The chronic recurring fits date from the onset in about a third of the cases. In the rest there is an interval before the recurring fits commence, which is usually at least a year, and may be fifteen or twenty years. It is not uncommon for the hemiplegia to occur in infancy, and the convulsions to commence at the epileptogenic period of puberty; sometimes a distinct exciting cause can then be traced. When the attacks date from the onset, as a rule this was attended with convulsions, but the converse does not hold good. When the hemiplegia came on in adult life, a long interval is rare; the fits usually commence in less than a year.

The degree of the hemiplegia at the onset varies; it may be considerable or slight; in young children, when trifling, it may be overlooked, the more readily if the child is gravely ill. Still more various is the degree of paralysis which persists. The leg has usually recovered, to a large extent or altogether. The arm is more often weak, sometimes powerless and small; occasionally it presents little or no evidence of the initial weakness. In half the cases the hand presents a state of "mobile spasm," in slight or considerable degree (see p. 86), and it is important to look for traces of this in doubtful cases, since it may be distinct when the weakness is not.

The convulsive attacks, in the majority of cases, begin in, and are confined to, the paralysed side. In rare cases, in which the attacks are severe, they may be always general. A distinct warning is present in about five sixths of the cases, far more frequently than in idiopathic epilepsy; and in half the cases it consists in a deliberate commencement of the attack in some part of the paralysed side. The other less frequent warnings are for the most part similar to those of the idiopathic form. The spasm is usually also of the same character.

Minor attacks are frequent in these cases, sometimes similar to those of idiopathic epilepsy (a faint, sudden giddiness, &c.), but more frequently they consist of the aura of the severe fit, often without loss of consciousness. Hysteroid seizures are also common in these cases, and so is considerable mental defect.

The Complications of epilepsy are chiefly the conditions that induce it, when they persist; these have been mentioned in the account of its cause: secondly, its effects on the nervous system and general health, especially associated hysteroid disturbance. Migraine and epilepsy are occasionally associated, sometimes in apparent independence, while sometimes the migraine ceases when the fits begin. In one remarkable case of this character the preliminary sensory disturbance of typical migraine persisted, in briefer form, as the aura of characteristic epileptic fits.

The Varieties of epilepsy have, for the most part, been mentioned in the account of the symptoms. The most important classes are the idiopathic and organic forms, the latter including post-hemiplegic epilepsy and epilepsy from active irritant brain disease. By some, the organic forms are termed epileptoid convulsions, but they may become established as an ineradicable disease, through the effect of the repeated discharges, to the idiopathic form.

Besides these, patients sometimes suffer from sudden brief symptoms, bearing more or less resemblance to some element of the minor epileptic attack, such as sudden transient causeless vertigo, or recurring causeless faints that have the aspect of cardiac faints. Many of these cases are probably on the border line of epilepsy, into which some would pass unless relieved. There is, indeed, some reason to think that cardiac faints, often occurring, may establish in the brain a tendency to pass into a state of similar functional abeyance—essentially *petit mal*.

Course of Epilepsy.—The disease may commence by severe fits or by slight ones. The latter may exist alone for months or years, and their nature may be unsuspected until a severe fit occurs. The interval between the first and second severe fit is sometimes prolonged, and is a point of considerable importance. When a single convulsive attack has occurred, the anxious question—whether it is epilepsy?—can only be answered by the occurrence or absence of other fits. In one third of the cases the interval is less than a month; in another third it is between one and twelve months; in the remainder it is more than a year, and it is occasionally ten years. Thus the chance of recurrence does not materially lessen until a year has elapsed.

When the disease is established the interval between severe fits varies greatly. It is less than a month in three quarters of the cases; in about half, it does not exceed two weeks; in about a tenth, fits occur daily. Usually the intervals vary, and they correspond to our current divisions of time only in the cases (not frequent) in which attacks in women are related to the menstrual periods.

The attacks may be isolated or grouped; the former is more common. When grouped, several attacks occur together, and then there is an interval of freedom. The number in each group varies from two or three to twenty. The patient usually recovers consciousness between each. In rare instances a series of fits occurs in which the patient does not recover consciousness; during the coma, one fit after another comes on. This has been termed the *status epilepticus*, and is a very grave condition. In its most severe form, the intervals between the fits become shorter, the coma deepens, the pulse and respiration become very frequent, and the temperature rises, it may be to 105° or 107° (Bourneville). The patient may die in a state of collapse, from the violence of the convulsions, or, the fits ceasing, he may become delirious, and present symptoms of meningitis, with rapid formation of bedsores, and may die in this stage. At any period the symptoms may lessen, and the patient recover. Many cases, however, end fatally, but fortunately the condition is rare.

Minor attacks occur in less than half the cases of epilepsy. They may occur alone, without severe fits, but both kinds are usually associated. The slight seizures are, as a rule, frequent. In more than half the cases in which they occur at all, there are daily attacks, usually from two to twenty each day, and I have even known as many as two hundred to occur every day. When there are no other attacks, the minor fits are usually frequent, and daily. They may also occur daily when there are severe fits, but when the minor attacks are less frequent, the patient usually has also severe fits. When the latter are frequent, minor attacks are often less frequent. Now and then, when the interval between the severe fits is more than two weeks, the minor attacks occur for a few days before (less commonly after) the severe fits.

Epileptic fits may come on when the patient is asleep or awake, or in both conditions. They occur in both, or in the waking state only, twice as frequently as in the sleeping state only. Very rarely the patient has them only in the act of going to sleep or of waking up. Now and then they are confined to the early morning. When fits that have occurred only during the night, occur during the day, they usually continue during the night, but if they have previously been diurnal only, and commence in the night, they very often cease during the day. Attacks which have occurred both day and night often cease in the day and continue in the night, but very rarely cease at night and continue during the day.

In women, attacks often occur at the menstrual period, although when closely investigated the relation is not found to obtain in more than about half the cases. The usual relation is for the attack to occur before, or (rather less frequently) during the period, rarely after it. The relation does not seem to be connected with any abnormal state of the uterine organs.

Death is a rare consequence of epilepsy, especially when allowance is made for the frequency of the disease, and the alarming aspect of the fits. When it does occur, it is scarcely ever from the direct effect of the convulsion, except in the "status epilepticus." It usually results from some accident to which the fit leads, such as a fall into the water or a burn. It may also be due to suffocation produced during the state of coma, either by vomited food getting into the larynx, or by the patient in bed turning over on the face.

PATHOLOGICAL ANATOMY.—The naked-eye appearance of the nerve-centres in epilepsy is, for the most part, that of healthy organs. In cases of long duration there is occasionally slight opacity and thickening of the meninges, and after death, in the *status epilepticus*, there may be signs of meningitis, but these are apparently merely secondary changes. If the patient has died in a fit, the post-mortem usually shows signs, in all organs, of the intense venous engorgement which is so conspicuous during life, and small extravasations of blood may be found, such as are met with in all cases of asphyxia. Of the histological changes revealed by the microscope, most of those which are not common, apart from symptoms, are apparently of secondary origin, the result and not the cause of the convulsions; and the various changes that have been described, and have been supposed to be related to the origin of the disease, have probably no connection with it, and do not even deserve enumeration. No greater significance can be ascribed to the induration of the cornu ammonis (pes hippocampi), to which weight has been attached by Meynert. Nor is there at present much likelihood that our knowledge will be more definite. The changes in the nerve-elements are probably of that molecular character which is revealed only by altered function, and eludes the most minute research.

PATHOLOGY.—In the absence of any help from anatomy and histology the pathology of idiopathic epilepsy is a matter of hypothesis, based on the influence of organic disease in causing similar symptoms, on the results of experiments on animals, and on the indications afforded by a study of the symptoms in the light of cerebral physiology.

It may be premised, as admitting of no question, that the muscular spasm is to be regarded as the result of the sudden over-action ("discharge") of nerve-cells, the violent liberation of nerve-force, and that the sensations which, in some cases, the patient experiences before losing consciousness must be due, directly or indirectly, to the same cause (see p. 737). The problem is, how this over-action is brought about, and where it occurs.

What is the primary seat of discharge? Experiment teaches that irritation of both the cortex cerebri* and the medulla oblongata† may cause convulsion, although it is doubtful how far the latter gives rise to convulsions resembling true epilepsy.‡ The teaching of pathology is, that disease which excites convulsions is most frequently at the cortex, and that whenever organic disease causes convulsions that begin locally, the disease is almost invariably at the cortex. In idiopathic epilepsy the convulsions sometimes begin in this manner, and this suggests very strongly that in such cases the process occurs in the cortex.

A further study of the mode of onset throws some light on the question, because the character of the aura indicates the function of the part in which the discharge commences. The auræ that consist in a disturbance of the function of the special sense centres, indicate that the discharge commences in those centres,—that is, in the case of smell and vision at least, within the cerebral hemispheres. In all cases in which organic disease causes convulsions so commencing, the disease has been in or close to the cortex (see p. 92). The warnings which consist of an intellectual process or an emotion, also point to the cortex as the seat of the primary discharge. Thus the significance of all the facts regarding the modes of onset that we can interpret, is that the discharge in epilepsy begins in the cortex of the cerebral hemispheres. The conclusion is confirmed by rare cases in which a lesion interrupting the internal capsule occurs in a person subject to general epileptic fits. The effect is that the fits, which before were general, almost cease on the paralysed side.* Such cases show that the discharge causing the general convulsions occurred above the internal capsule, *i. e.* in the cortex of the brain. If there is evidence that so many cases of idiopathic epilepsy depend on discharge of the grey matter of the cortex, it is probable that the conclusion is true of all cases. The wide variety of auræ seems to show that the primary discharge may occur in various parts of the cortex. The fact that the first discharge may be uniform in character in the same case—may be such, for instance, as to cause a definite and even complex sensation—can only be explained by the theory that the derangement of function in the nerve-cells is inherent in them, and is not the result of any random influence outside them.

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* Ferrier, Luciani, Bartholow, Horsley, &c.

† Brown-Séquard, Kussmaul, Nothnagel.

‡ Binswanger ('Arch. f. Psych.,' 1888) found that, in animals, tetanoid attacks of spasm in the limbs and trunk could be produced by electrical stimulation of the lower half of the floor of the fourth ventricle on each side, and concluded that they were reflex from a centre in the dorsal and upper region of the pons; but he never succeeded in causing epileptic attacks of the common type.

All parts of the brain are intimately connected, and the *sudden*, *i. e.* instantaneous, derangement of the function of any part appears incompatible with the integrity of consciousness. This is shown by many facts of organic disease. Hence it is intelligible that sudden discharge, wherever it occurs, may cause loss of consciousness. But if there is reason to believe that sudden discharge may occur in any part of the grey matter of the cortex, it may occur in that which subserves directly the phenomena of consciousness, and a sudden alteration in the functional state of these structures may well be, as Hughlings Jackson has suggested, the most probable explanation of the attacks which consist only of such momentary loss.

It is necessary, however, to allude briefly to other theories which have been, and are still, current regarding the nature of epilepsy. It has been widely held that the morbid action in the brain is excited by arterial spasm, causing cerebral anæmia, and also that the convulsions originate from the primary discharge of a convulsive centre in the medulla, but that the loss of consciousness is produced by vaso-motor spasm in the brain. In each theory it is assumed that the vaso-motor spasm is due to the sudden over-action of the vaso-motor centre in the medulla. The evidence on which these theories are based is that cerebral anæmia will cause loss of consciousness and convulsion,[*] and that at the onset of epileptic fits there is always pallor of the face. But the latter statement is, as we have seen, incorrect. If it were true, it would not justify the conclusion drawn from it. We do not infer that the brain is congested whenever the face blushes, and why should we assume that the brain is anæmic when the face becomes pale? There is no necessary correspondence between the condition of vessels in the skin and in the viscera beneath, and probably even less in the case of the brain than in that of other organs. It is far more probable that the spasm of the vessels of the face is the *result* of the cerebral discharge, just as the arteries of a frog's foot will contract when its brain is irritated. In rare cases in which an early failure of the heart occurs, this may also be the effect of the discharge, which, as it often influences consciousness first through the central connection of the pneumogastric (see p. 738), may also affect the periphery by the same channel. The arteries of the brain may possibly be thus influenced in a secondary manner.[†] That sudden anæmia of the brain will cause convulsions is no proof that the fits of idiopathic epilepsy are so produced. Such a theory can give no explanation of the complex character of the commencing discharge, or of its uniformity in the same patient.

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* Oebeke has recorded such a case, and I have met with a similar instance, although unconfirmed by autopsy.

Thus the vaso-motor theory of epilepsy is alike unneeded, unproved, and inadequate. The phenomena indicate that there is discharge of grey matter, and there is nothing to warrant us in going beyond the grey matter concerned, in our search for the origin of the discharge; it is certain that this may commence in various parts of the cerebral cortex, and there is no evidence that it is dependent upon any antecedent.

Can we form any opinion as to the nature of the change in the grey matter which permits the sudden liberation of nerve-force? It is necessary to remember that we have direct evidence only of *liberation* of force, but we must recognise, in all nerve-cells, a function by which the liberation of energy is restrained, a resistance to action as well as a capacity for action.[‡] Of the nature of this resistance we can form no idea, but we cannot conceive of the function of cells without it, and the idea has underlain such expressions as "nerve tension" and the like, which have been long employed. The phenomena of epilepsy suggest that the instability of the grey matter, its tendency to discharge, depends on instability of resistance, rather than on any primary change in the energy-producing action of the cells, although the latter normally in constant activity may be secondarily augmented by the increased demand. Analogy suggests that the internal resistance to action is a higher function of the cells than the production of force. Hence the fact that there is over-action is consistent with the evidence that there is imperfect nutrition. Moreover, the view that it is the resistance which is unstable enables us to understand the phenomena of inhibition which sometimes occur as part of the attack. In a slight degree of disturbance the resistance may be suddenly raised instead of being lowered. It is possible that the attacks in which there is loss of consciousness only, may be thus produced,—may be analogous to the fits in which there is sudden darkness rather than to those in which there is a flash of light.

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[*] Kussmaul and Tenner.

[†] Hume observed that the pulsation of the brain, in the opening left by trephining for traumatic epilepsy, ceased during the fits, and that the brain became less prominent ('New York Med. Rec.,' 1889).

[‡] Handfield Jones, Michael Foster, Ringer, &c.

DIAGNOSIS.—The first point in the diagnosis of epilepsy is the recognition of the occurrence of attacks. Convulsive attacks, which occur in the daytime, are never overlooked, but if an attack occurs during sleep, and its onset does not awaken the patient, he may sleep on when it is over, and be unaware of its occurrence. Often there are some subsequent indications of what has happened. The tongue may be sore, there may be a little blood on the pillow, an extravasation beneath the conjunctiva, ecchymoses on the face, or a severe headache. These (except the last) are of unequivocal significance, but the patient may be unaware of their occurrence, and nocturnal attacks may occur for twenty years without the patient or his friends suspecting the fact. Minor seizures are also often unrecognised, not because they are unnoticed, but because they are not thought to be of any significance or serious nature. Those that consist only in a subjective sensation may not be mentioned by the patient. It is usually sufficient to be aware of their common forms, and to inquire for these, in order to ascertain their occurrence.

Certain forms of minor attack may, however, be confounded with other paroxysmal symptoms of different nature. The simplest form, in which there is merely brief unconsciousness, is not only called a "faint," but is often supposed to be actually syncopal in nature. The distinction from syncope rests, first, on the absence of obvious exciting influences, such as cause ordinary fainting. Epileptic faints often occur when the patient is sitting still, in a cool room, under no excitement. Consciousness is lost more suddenly in epilepsy than in syncope. The latter is usually preceded by a sensation of faintness, but so also, sometimes, are the minor seizures of epilepsy. The presence of this, and of vague "dizziness," palpitation of the heart, nausea, and cephalic sensations other than pain, is of little diagnostic significance. On the other hand, the absence of any warning sensation, or the occurrence of a distinct warning other than one of those just mentioned, is in favour of the epileptic nature of the attack. A sudden return of normal consciousness is in favour of epilepsy. If a normal condition is slowly regained, there is physical prostration in syncope, mental confusion in epilepsy, and in the former the pulse at the wrist is often scarcely perceptible. The passage of urine during the attack, muscular spasm, however slight, and automatic action or deep sleep after the attack, are certain proof of its epileptic character. In many cases the patient has had other more severe seizures which assist the diagnosis.

The attacks characterised by vertigo have to be distinguished from other forms of sudden giddiness. The latter are scarcely ever attended by loss of consciousness, and are usually followed by long-continued vertigo, which persists while the patient is recovering, and slowly passes away. In epilepsy there is generally loss of consciousness, and the patient is either quickly well, or is a little "dazed" after the attack; the vertigo may be associated with some other warning sensation, and there is often micturition. The vertigo most likely to be confounded with epilepsy is that connected with a morbid action of the auditory nerve, "labyrinthine vertigo." In addition to the diagnostic indications just mentioned, there is usually, in this form, slight persistent giddiness in the intervals, persistent tinnitus aurium, and some deafness. In epileptic vertigo there may be an auditory sensation, with giddiness, as the aura of the attack, but there are no persistent symptoms. It must not be forgotten that auditory vertigo and epilepsy may be associated; I have seen several instances of this.

If a patient suffers from distinct convulsion, the next question is, are the attacks epileptic or hysteroid? If an attack can be witnessed, the nature of the convulsion is usually obvious. The violent tonic spasm and shock-like clonic spasm of the typical epileptic fit, with complete unconsciousness and with cyanosis, and also the brief duration of the attack, are wholly unlike the prolonged tonic contraction, opisthotonos, wild co-ordinated movements, quick clonic spasm, perverted mental state, talking, biting, and convergent strabismus of the hysteroid seizure. More difficulty, however, may be presented by the untypical forms of epileptic fits, especially by those which consist only of tonic spasm. Evidence of their epileptic nature is afforded by the brevity of the attacks, their suddenness of onset, their occurrence apart from emotion and when the patient is alone, and the absence of hysteroid symptoms. When an attack has not been witnessed, and the diagnosis has to be made from the description of the patient or friends, it is a much less easy task. The chief diagnostic indications between pure epileptic and pure hysteroid fits are given in a tabular form on the next page. Especial care should be taken, in asking about the character of the convulsion, to avoid a leading question, and if suggestions are indispensable, to put them alternatively, thus: "Should you say that the patient *struggled* or *jerked* during the fit?"

If there is evidence that the visible convulsion is hysteroid in character, we have still to ascertain whether it is primary or is consecutive to an epileptic seizure, and this is often most difficult. When the initial epileptic fit is severe, its occurrence can usually be ascertained, and tongue-biting alone may be taken as establishing its occurrence.[*] It is when the epileptic attack is of the minor form that the diagnostic difficulty arises, because the initial stage of many hysteroid fits has a pseudo-epileptic aspect. The most important guide is the fact that most patients in whom the hysteroid attacks are post-epileptic, have at other times, or have had in the past, epileptic fits, major or minor, without this sequel, and the character of these corresponds to the commencement of the compound attack. In many cases the characters of the initial stage include some feature, such as micturition, which is conclusively epileptic. In most patients, moreover, who have had recurring attacks during several years, these are epileptic, and the hysteroid convulsion is secondary. The fact that the patient presents other symptoms of hysteria should be allowed no weight until all indications of epilepsy have been excluded, because, as already stated, it is only in those who are in some degree the subjects of hysteria that the combination occurs. It must not be forgotten, also, that separate hysterical and epileptic attacks sometimes occur in the same individual.

	EPILEPTIC.	HYSTEROID.
Apparent cause	none	emotion.
Warning	any, but especially unilateral or epigastric auræ	palpitation, malaise, choking, bilateral foot aura.
Onset	always sudden	often gradual.
Scream	at onset	during course.
Convulsion	rigidity followed by "jerking," rarely rigidity alone	rigidity or "struggling," throwing about of limbs or head, arching of back
Biting	tongue	lips, hands, or other people and things.
Micturition	frequent	never.
Defæcation	occasional	never.
Talking	never	frequent.
Duration	a few minutes	more than ten minutes, often much longer.
Restraint necessary	to prevent accident	to control violence.
Termination	spontaneous	spontaneous or induced (water, &c.).

If the attacks are recognised to be epileptic in character, before we can refer them to idiopathic epilepsy we have to ascertain that they are not due to reflex irritation, to toxæmia, or to organic brain disease. The irritation of dentition, intestinal worms, and, occasionally, indigestible food, are the most frequent causes of fits that may be confounded with those of epilepsy. In all cases in which the convulsions are of recent origin, these causes should be sought for and excluded, if necessary, by treatment. There is no other way of avoiding error, since the indication derived from the character of the attacks is an uncertain criterion. It must be remembered that fits may be at first of reflex origin, and may persist, when the peripheral irritation is removed, as idiopathic epilepsy. It is most unlikely that fits that have continued for more than a year are still due to reflex influences.

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[*] It is said that French hysterics bite their tongues during the attacks. In this country tongue-biting is practically confined to epilepsy.

Convulsions that are due to blood-states (alcohol, lead, uræmia) are rarely confounded with those of epilepsy, because they are merely part of a group of symptoms of obtrusive significance. The importance of an examination of the urine in all doubtful cases need not be insisted on. The recurring fits which may occur in chronic Bright's disease without other signs of uræmia, and in some cases of lead poisoning, may resemble epilepsy very closely, and are to be distinguished only by the discovery of the underlying condition. A mistake in diagnosis is especially probable in cases, previously unknown, in which albumen is present in the urine after an attack. But the absence of other indication of chronic and advanced renal disease should prevent a mistake.

The problem of the diagnosis of epilepsy from the convulsions of organic brain disease presents many aspects, and is of great importance. Those convulsions which attend a sudden acute cerebral lesion are not likely to be mistaken for epilepsy, nor is the converse mistake probable, except during the transient "post-convulsive" weakness after a first unilateral fit. Chronic brain disease, however, may cause convulsions, readily mistaken for those of epilepsy. Those convulsions usually begin locally, and may be partial in range. This character, while it suggests, does not prove, that they have this origin, since the convulsions of idiopathic epilepsy may commence in a similar manner, and, moreover, the convulsions of organic disease may not begin locally, but may be at once general. For the diagnosis, a careful search must be made for other symptoms of organic disease, —persistent headache, permanent hemiplegic weakness (not merely after an attack), paralysis of cranial nerves (especially diplopia), vomiting, and optic neuritis. The importance of an ophthalmoscopic examination in such cases cannot be too strongly insisted on. In any case of doubt, causal influences may be allowed weight. A history of syphilis, which so often causes cortical disease and convulsions, on the one hand, or a family history of epilepsy or insanity on the other, may rightly turn the diagnostic scale.

Convulsions may be due not only to active brain disease, but to the influence of an old cerebral lesion, an atrophied tumour, and especially a spot of old softening. In the former case the diagnosis rests on the history of the early symptoms. The latter are the cases of post-hemiplegic epilepsy already described. The distinction of these from ordinary epilepsy is easy if distinct hemiplegia persists, but may be difficult if this has passed away. The difficulty arises only in the cases which date from childhood. In all such cases, if the fits are unilateral, careful search should be made for signs of weakness or of spasmodic over-action. Slight traces of the latter are significant. The circumstances of origin should also be ascertained. In infancy trifling hemiplegia is readily overlooked, and if the first convulsions were severe and unilateral, corresponding in seat to those which still occur, the case is probably post-hemiplegic, even though no hemiplegia was noted, and although an interval of years elapsed between the initial convulsions and their recurrence. Epileptoid fits may be an early symptom of general paralysis of the insane, but the tremor of lips and tongue, the unequal pupils, and the menta change render the diagnosis easy in most cases.

The simulation of an epileptic fit is on the whole rare, and the pretended fit never closely resembles a genuine seizure. In all cases the perfect reaction of the pupil to light throughout the fit will be conclusive evidence. Too much weight must not be placed on the untypical character of the attack, because true epileptic fits may deviate much from the usual type.

Prognosis.—The risk to life in epilepsy is not great. The mere violence of the fit, appalling as may be its aspect, rarely causes death. The dangerous "status epilepticus" is too exceptional to constitute a measurable element in the prognosis. The greatest danger is in the cases in which there is a tendency to turn on the face, or to vomit after a fit, but even this is slighter than that of the accidents to which the attacks expose the patient. Many epileptics die by drowning; the fit not only occasions the fall into the water, but prevents any effort to escape, and hence an epileptic has more than once been drowned in a ditch.

The prospect of a spontaneous cessation of the fits is small; the tendency of the disease is to self-perpetuation. Occasionally convulsions, during infancy, cease at four or five years of age. Attacks which have continued till puberty rarely cease at that epoch. After twenty, spontaneous cessation does sometimes occur, and I believe that it becomes more frequent as life advances, but it is too rare to be reckoned on.

The chief question, therefore, is—what is the prospect that the disease will be cured, or held in abeyance, by treatment? We cannot at present separate the two points, and the only way known of curing the disease is to keep the fits away for a sufficient length of time to permit the morbid tendency to subside. Hence the question resolves itself into the probability of complete arrest. The prospect of arrest is slightly better in males than females, better if the disease begins after twenty than before, and better the shorter the duration of the disease, being greatest in the cases in which it has existed for less than a year. It is rather easier to arrest the fits when there is an hereditary tendency than when there is not—a curious fact, which was also pointed out by Herpin. The presence or absence of an exciting cause for the first fit does not influence the prognosis. A longer interval between the fits increases the prospect of arrest; this is extremely rare if fits occur daily. But this consideration is interfered with by the great difficulty of getting patients, whose fits occur at long intervals, to persevere with treatment. The prognosis is better if the fits occur only during the sleeping or the waking state, than if they occur in both. It is better if there is no considerable mental change, and if the attacks are all of the severe variety than if there are minor seizures, and better if the attacks are preceded by an aura than if they occur without warning. In cases of post-hemiplegic epilepsy the prognosis is much less favorable than in the idiopathic cases.

TREATMENT.—The treatment of epilepsy consists partly in the general management of the patient, and partly in the administration of drugs to influence the attacks,—to arrest their occurrence, or, failing this, to render them less frequent and less severe. Unfortunately, the influence of all drugs is transient, and has to be repeatedly renewed. No means is known of suddenly curing the disease, of suddenly effecting such a change in the nerve-centres that the attacks do not again occur, nor are there at present any facts which render it likely that such means will be discovered. The only method of producing such a change is by the continued administration of drugs for a long time, so as to prevent the occurrence of the nervous discharge, and thus to produce such a change in the cells that the medicine may ultimately be discontinued without a recurrence of attacks. It is of great importance that this need for prolonged treatment should be made clear to all patients, and its reason to all those who can understand it.

Since the introduction of bromide salts for the treatment of epilepsy they have superseded other drugs to a large, and some think a too great extent. In the majority of cases their influence is incomparably greater than that of any other remedies, but in a minority of cases they fail, and in some of these other agents are more powerful. They only do permanent good by continued administration. The absence of a permanent effect from a short course of treatment is, however, equally conspicuous in the case of other remedies. Bromides are said to cause contraction of the small arteries of the brain, but it is exceedingly doubtful whether any part of their influence in epilepsy is due to this action. The various effects of their administration suggest strongly that they have a direct action on the nerve-cells. On any theory of epilepsy we must ascribe it ultimately to the disturbed action of nerve-cells in some situation, and it is unnecessary to go beyond this influence of bromide on nerve-cells to explain its action.

If we regard the morbid state in epilepsy as an instability in the resistance of nerve-cells, it seems probable that the effect of bromide is to increase the stability of that resistance.

The bromides of potassium, sodium, and ammonium are extensively used, and bromide of lithium is occasionally employed; some authorities express a preference for one, some for another, some for a combination. My own experience has been that the influence of each on the disease is nearly the same, but that the bromides of sodium and lithium are rather less effective than the others, and that the bromide of potassium is a little better borne than the bromide of ammonium. It is probable that very little, if any, of the bromide salt is decomposed in the system, and that the base does not exert its special influence. Bromide of gold, of nickel, and that of ammonium and rubidium have been tried, but the evidence of their value is inconclusive. The administration of free bromine has been suggested, but it is difficult to give it in sufficient quantity, and it must be transformed into bromide as soon as it enters the alkaline blood. This is true also of hydrobromic acid. No salt of bromine has much less tendency than another to produce acne, and this can always be prevented or rendered extremely slight by giving arsenic at the same time. Periodical omission does not prevent the rash.

Bromide is usually given continuously in the smallest doses which will arrest the fits, or, failing this, in such doses as produce the most marked effect upon them. When the fits occur at a certain time, one daily dose may be given two or three hours earlier. If the attacks occur at various times the bromide must be taken two or three times a day. The total daily quantity may vary between fifteen grains and two drachms, according to the age of the patient and the effect of the drug. Few patients are able to bear more than a drachm and a half a day without becoming what is termed "bromised," lethargic and dull, physically and mentally, with cold extremities, and a feeble pulse; the best results are usually obtained with not more than a drachm a day. If this does not arrest the attacks, larger doses rarely succeed, and combinations of bromide with other drugs are more useful. The effect of bromide is sometimes immediate: after the first dose the attacks may cease; often, however, its influence is gradually produced. When the attacks have ceased under its administration, they too often relapse if it is discontinued, and then are less easily arrested than at first. Relapse usually occurs within a few weeks (sometimes within a few days) of a too early cessation of treatment; after a year of freedom without treatment it is probable (though not certain) that the disease will not recur. As a rule, bromide should be continued, without any diminution of the dose, for two years after the last fit. It should not then be suddenly discontinued, but the daily dose should be gradually lessened through another year. The continued use of bromide in moderate doses has no necessary influence on the general health or intellectual energy. At first, however, some cerebral depression may often follow the arrest of fits (see p. 749), and this is often erroneously regarded as the effect of the bromide alone. If moderate in degree it is better to combat it by tonics than to reduce the bromide. Occasionally the depression becomes so alarming that it is necessary to stop or lessen the medicine, and even to allow a fit to occur, and to go on with smaller doses.

In order to facilitate the occurrence of the change in the nutrition of the nerve-cells which we must assume to underlie the cure of epilepsy by bromide, I have found it well, in cases in which a moderate dose stops the fits, to administer a series of large doses at increasing intervals, beginning with two drachms every second morning, and increasing to three drachms every third morning, and four every fourth;* the dose and interval are then reduced in the reverse order, so as to spread the course over about six weeks. The dose should be given after breakfast, in about half a pint of water. After such a course, if the bromide is discontinued, patients remain free from fits much longer than after bromide has been given for the same time in ordinary doses, showing that more effect on the nerve-elements has been produced. But a permanent result is seldom obtained from such a course alone; it is still necessary to continue small doses for a year or more. I believe, however, that the prospect of cure is increased by this cumulative treatment.

When bromide alone fails, it may succeed when combined with certain other drugs, most of which have by themselves some influence on the disease. One of these is digitalis, a popular remedy for epilepsy in the west of England two centuries ago. The combination is useful, as might be expected, when there is cardiac dilatation and valvular disease, but it is also specially useful in nocturnal epilepsy, and in some other cases. Digitalis probably has an action on the central nervous system, as well as on that of the heart and vessels, although some part of its influence may be due to the regulation of the blood-supply. Five or seven minims of the tincture may be given with each dose of bromide. Another combination of value is that with belladonna, which is also an old remedy; and, although very rarely successful alone, it is a useful adjuvant, in doses of five or ten minims of the tincture. Atropine may be given instead, one or two drops of the B. P. solution. The combination of bromide treatment with the occasional passage of a voltaic current from the hand to the opposite side of the skull, over the motor region of the cortex, would scarcely deserve mention, had it not received the surprising commendation of Niemeyer.

Cannabis indica is occasionally beneficial, both alone and in combination with bromide; the combination is most useful in cases with a good deal of persistent headache. Opium and its alkaloid morphia are of little service in epilepsy. The hypodermic injection of morphia in full doses is attended with considerable danger. If an attack occurs after the injection has been given, and the post-epileptic coma coincides with the narcotism, the patient's life may be in great danger, and I have known death to occur, apparently from this cause.

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* I have several times increased the dose up to an ounce every five days (more is usually vomited), but slight mental derangement, lasting for a few weeks, is apt to be set up. The chief immediate effect of a dose of four to eight drachms is head ache

Zinc has long been held in repute, and with some reason. It is far inferior to bromide in most cases, but now and then succeeds when bromide fails. The lactate of zinc, introduced by Herpin, is the most convenient form; it is the most soluble of the less irritant salts of zinc, and if given after meals it can generally be increased to eight or ten or even fifteen grains, twice or three times a day, without producing nausea. The oxide of zinc may be given if the lactate is not accessible, but the limits of toleration are sooner reached. The citrate answers almost as well as the lactate. The bromide of zinc is also a useful form. Either may be combined with belladonna or with bromide of potassium, and the combination of the three sometimes succeeds when each alone fails.

Iron, in the opinion of some distinguished authorities, should not be given to epileptics, because it is thought to increase the frequency and severity of the fits. This opinion, as a general principle, is certainly erroneous. I have given iron to several hundred epileptics, and instances of apparent aggravation of attacks are extremely rare. In most cases it may be given without any ill effect on the disease, and in some its use is distinctly beneficial. I have known attacks to cease entirely when iron was added to bromide, and, in rare cases, when iron was substituted for bromide. Care must of course be taken not to ascribe to the iron any effect of the discontinuance of the bromide. Iron seems to have a direct action on the nerve-centres, analogous to that of zinc.

In some cases of inveterate epilepsy, in which bromide had no influence, I have found borax distinctly useful. From fifteen to thirty grains may be given after food three times a day, and it may be continued for years without any ill effect beyond a possible eruption of psoriasis, amenable to arsenic. A little gastro-enteric disturbance may occur at the commencement of administration, but quickly ceases if the dose is lessened. Of course the influence of borax is not comparable to that of bromide in cases in which this is effective.*

Nitro-glycerine is sometimes useful, especially for the minor attacks. It may be given in doses of $\frac{1}{200}$ of a grain to begin with, increased gradually to $\frac{1}{75}$, half a minim to $1\frac{1}{2}$ minims of the Tinct. Trinitrini (of the B. P. Supplement) after food, three times a day. In one case, with rather severe fits, these became fewer as the dose was increased, and finally ceased when the patient was taking $\frac{1}{80}$ grain, and did not recur. It often prevents the occurrence of the post-epileptic hysteroid convulsion. The alcoholic solution is the best form, and may be combined with bromide, if a few drops of hydrobromic acid are added. Among other drugs which I have tried without seeing benefit, other than slight and rare, from their use, are aconite, hydrocyanic acid, bromide of camphor, nitrite of soda, nitrite of amyl (by the mouth), chloral hydrate, paraldehyde, benzoate of soda, Piscidia erythrina, Calabar bean, ergot, sclerotic acid, codeia, and cocculus indicus. It is singular that the last, injected beneath the skin, will infallibly produce a fit in an epileptic patient. From nitrate of silver I have seen little benefit, and I have had, among my patients, several who were discoloured by it, in the prebromidic days, without any alteration in the severity of the attacks. Antipyrin (25 grains daily) is said to have arrested attacks in a child of three.*

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* The use of borax was, I believe, first recommended in my Gulstonian lectures on Epilepsy (1870). Its value in cases that resist bromide has been amply confirmed by Folsom, Jones Hill, Stewart, and others in America, by Mairet in France, and by Spencer, Russell and Taylor, and others in England.

The treatment of minor attacks is on the whole the same as of the severer seizures. They are often arrested by bromide, but it is far more common for bromide to have no influence in the case of *petit mal* than in convulsive attacks. The latter may be arrested, and the former may continue or even become more frequent. The other drugs above mentioned are sometimes effective when bromide fails, especially the salts of zinc, belladonna, and Indian hemp. Bromide of zinc has been already mentioned; it may be given in doses of two to four or five grains after food, well diluted, and combined with alkaline bromides in cases in which both forms of attack occur. There is much variation in the individual gastric tolerance of zinc; it can often be better borne with $\frac{1}{12}$ gr. of cocaine hydrochlorate. Ethylene bromide has been said to lessen minor seizures, but not to arrest them.

Arrest of Attacks.—The means by which commencing fits may be arrested have been already mentioned. In attacks commencing in one extremity, the ligature is often successful. The most convenient method of applying it is for the patient to double a piece of tape, and pass it round the arm above the elbow, with the ends through the loop formed by the doubled part, and brought down to the lower part of the sleeve so as to be accessible, and easily pulled tight, as soon as the warning is felt. Now and then the repeated arrest of fits produces a permanent effect: in one patient, for instance, the aura ultimately stopped spontaneously at the place where it had been many times arrested by the ligature. In cases of this kind an attempt has been made to produce a more permanent effect by a blister around the limb.† It is occasionally successful; but the arrest of the fit, by the ligature or a blister, sometimes causes so much giddiness and distress that some patients have considered the remedy worse than the disease.

Of methods of arresting fits which begin in other ways, inhalation of nitrite of amyl is that most frequently (though not invariably) successful; it doubtless acts by flooding the brain with arterial blood, a potent agent for modifying the action of the nerve-elements.

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* Anderson, 'Am. Journ. Med. Sc.,' 1891.

† *An old method of treatment,* revived by Brown-Séquard and Buzzard.

During an attack little treatment is necessary. In patients who bite the tongue, a cork or, better, a small piece of india-rubber placed between the teeth will prevent this accident. The patient should be laid down, for obvious reasons; it is doubtful, however, whether posture influences the duration or severity of the attacks. It is necessary to see that the clothes are loose about the neck; if they are tight when the neck becomes turgid and swollen, the resistance to the return of blood is increased, and extravasations into the skin and conjunctivæ are more probable. After the attack, the patient should be allowed to sleep, if inclined to do so, for at least half an hour.

In the *status epilepticus* bromide often fails. The inhalation of chloroform usually effects only a transient amelioration. Nitrite of amyl has been recommended by Crichton-Browne. In the cases I have seen, most influence has been exerted by chloral (gr. xv every three or four hours), subcutaneous injections of morphia (gr. $\frac{1}{10}$), and the application of ice to the spine.

General Management.—It has been recommended, on theoretical grounds, that the diet of epileptics should contain little or no animal food. The evidence of experience, as far as I have seen, is opposed to this opinion. I have known the exclusion of meat from diet to cause a great increase in the severity of the fits, which became slighter when meat was again given; and I believe that patients do best if a moderate quantity of animal food is given twice a day, care being taken to avoid that which is indigestible. I have known one case, however, in which the patient could never take beef without bringing on an attack, although he could take other kinds of meat with impunity. Stimulants should be taken sparingly; young persons do better without alcohol. In all cases it is most important that the regular action of the bowels should be secured.

Moderate exercise of body and mind does good, but severe and exhausting exertion is undesirable, and the excitement of competitive and other examinations should be avoided. The education of children should not be entirely neglected. It must be remembered that there are many positions in life for which epilepsy constitutes no insuperable disqualification, and for which they may be trained, should the disease not be arrested. In this choice of an occupation, so much depends upon personal opportunities that it is difficult to lay down general rules. But there is one consideration to which all others must be subordinate,—the calling must be one which involves no risk of life from the occurrence of an attack while the patient is at work. An outdoor life is better than sedentary occupations, but the choice of the latter is so much larger that in most cases a sedentary calling has to be selected.

The parents of a sufferer often conceal from him or her the nature of the malady, terming the seizures "faints," in order to avoid distress. Sometimes, however, it is impossible to secure the needful caution, and persistence in treatment, unless the nature of the malady is known. But it is wise for the practitioner to cultivate the habit of using the word "attack" rather than fit, lest he give inadvertently a grave shock to a patient who was before in ignorance.

The question of marriage presents itself under two aspects, as regards the individual and as regards the possible offspring. Marriage has no influence on the disease, beneficial or the reverse, except so far as it may involve deleterious sexual excess. But with reference to the offspring the question is very important. There is no certainty that the taint will be transmitted; on the contrary, as regards any individual child there is a probability that it will escape. But the probability is also against the escape of all the offspring from diseases of the nervous system, at least when the disease in the parent is inherited.

Surgical Treatment.—Counter-irritation in the neck or scalp has been often employed, the usual method being by a seton in the neck. That it occasionally does good is undeniable, and the same is true of an extensive accidental burn. In most cases the effect is temporary only. As the cases mentioned in the section on Etiology show, in the rare form of epilepsy in which the fits are excited by pressure on a tender cicatrix, or are preceded by pain at such a scar, it is right to excise the cicatrix, amputate a finger, or at least resect a nerve.

Trephining, an old remedy, has been lately brought into fresh prominence. The mere formation of a hole in the skull, without interference with the brain, has been employed in many cases of idiopathic epilepsy, but the results obtained are little better than those of a seton in the neck, and it is doubful whether the operation has other influence than that of an energetic counter-irritation. But the local commencement of the fit in one limb means local excessive instability in the corresponding cortical centre, and in some cases it means also old organic disease. (When there is active disease there is probably a tumour, and such cases have been already considered.) The question comes, Is the removal of such disease justifiable? The answer must depend on the character of the fits, and on their apparent cause. If many of these are local and partial, and there is reason to believe that there is disease that can be completely removed, an operation is justifiable. If, indeed, there is no evidence of removable disease, the excision of the centre, stimulation of which causes the movement with which the fits commence, may arrest them, and if the area excised is not very extensive, the loss of power it causes soon lessens to a moderate degree, as Horsley and others have abundantly proved. If, on the other hand, the fits generally spread, not only through the side on which they began, but also to the other side; if they begin with an aura such as is common in idiopathic epilepsy; or if the patient has also minor attacks similar to those of the idiopathic form, the probability of benefit is much diminished. Apparently the *repeated discharges* have led to so wide a deficiency in the stability of the nerve-cells that the discharge will start from other regions if the primary lesion is removed. This is the teaching of experience in a considerable number of such cases in which the operation has been performed, including some in which they were caused by the pressure of depressed bone or an exostosis, and the cause of the irritating pressure could be easily removed, as well as others in which the affected centre was excised. On the other hand, in both sets of cases, when the attacks always began by local spasm and remained one-sided, the operation has been successful, even after many years.*

Ligature of the carotid artery has been performed without result. Ligature of one or both vertebrals has been practised by Dr. Alexander, of Liverpool (and by others after him), as a cure for epilepsy, but the results obtained are not commensurate with the risk, although this is not so great as might be thought. In a few cases the attacks have ceased; in the majority they have returned after a period of a few weeks or months.

CONVULSIONS: ECLAMPSIA.

Convulsions, resembling more or less closely those of epilepsy, may occur from various causes. In epilepsy, however the disease was originally excited, the recurring convulsions are the result solely of the tendency of the brain to "discharge," and, beyond trifling disturbing influence, no cause outside the brain can be discovered. When such fits are due to some other cause, they are called simply "convulsions." But this term has also a wider application; it is applied to the fits of epilepsy, and also to those that are produced by organic brain disease. Hence the word "eclampsia" has come to be used as a name for the condition in which convulsions occur from other causes than primary states of the brain. In epilepsy, the convulsions themselves are the sole evidence of their cause; in eclampsia, the cause of the fits manifests itself by other symptoms, often by symptoms outside the nervous system.

The term "eclampsia" is, however, chiefly used as a designation for convulsions that recur. It is not applied to the single fit that a child may have at the onset of an acute fever, or in consequence of an indigestible meal. Moreover, the convulsive attacks that form part of the manifestations of hysteria are also excluded from the meaning of the term. There are three special varieties of eclampsia,—infantile, puerperal, and uræmic.

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* Such cases are within the province of surgery rather than that of medicine. Details of many cases of the kind will be found in the writings of Horsley, MacEwen, and others.

INFANTILE CONVULSIONS: INFANTILE ECLAMPSIA.

Convulsions occur in young children with great readiness, and have many causes. The special liability of infants is probably due to the condition of development of the nervous system. At the time of birth, only parts of it are structurally complete. Extensive tracts of fibres have not yet acquired their white medullary substance, and until the axis-cylinders are thus clothed, the fibres have but little conducting power, although it is probable that such power is not altogether absent. But the lower centres are farther advanced than the higher ones, and are, in consequence, imperfectly controlled. This is probably the chief reason why reflex disturbance so readily occurs in early childhood. By far the most potent cause of convulsions in children is the constitutional condition termed "rickets." The essential element in rickets is defective development; the perversion of development that occurs (*e.g.* in the bones) is secondary to, and consequent on, its defect. At the time at which this constitutional state chiefly occurs, the structural development of the nervous system is complete. But it is probable that functional capacity is only fully developed after structural perfection, and the parts last developed must suffer from the general delay in development more than those parts that have been longer perfect and longer in full use. The lower motor centres in the spinal cord, medulla oblongata, and even in the cerebral cortex, are under less than the normal amount of control; they pass into a condition of over-activity, are excited by peripheral impressions with undue readiness, and thence is produced the series of symptoms of excessive reflex action, laryngismus stridulus, carpo-pedal contractions, tetany, and convulsions. It is probable that the morbid tendency is exalted by an inherited neurotic disposition.

The period at which rickets chiefly occurs is between the sixth and eighteenth months of life. This period corresponds with the active epoch of dentition. Dentition is delayed, with other developmental processes, and hence the fits have commonly been ascribed to dentition, and have been called "teething fits." It is probable that, in some cases, the process of dentition has an influence; it involves irritation of the sensory nerves, and so may excite the convulsion. But it is certain that this exciting influence is only a small part of the process of causation, and that it was formerly over-estimated, even after the relation of the fits to rickets had been conclusively proved by Sir William Jenner. The convulsions may occur without any discoverable exciting cause, or they may be produced by various peripheral impressions besides those of the teeth, both in children who are rickety and in those who possess only the predisposition inherent in infancy. Of these causes the most frequent is some irritation of the gastro-intestinal mucous membrane, especially by worms or by some substance in food that is absolutely indigestible, such as the skins of dried fruit, currants, &c. Among worms, lumbrici are chiefly influential; tapeworms are rare in young children, and threadworms scarcely ever cause convulsions, although they are often thought to do so. The influence of irritation of the alimentary membrane in causing convulsions is remarkable; the effect often occurs with very little conscious sensation, while contractions of the intestines that cause acute pain have no corresponding influence.

Infantile convulsions may also be caused by morbid blood-states, and may attend the onset of many acute diseases. They also occur in states of general exhaustion, however these are produced, especially in that caused by diarrhœa. Thus they may form part of the condition called "hydrocephaloid" (p. 370). The predisposition in these cases probably depends on the acute impairment of nutrition of the nerve-elements, perhaps also on the deficient blood-pressure, of which the depressed fontanelle is evidence. On the other hand, they result also from mechanical congestion of the brain, which involves, among other conditions, a deficiency in the supply of arterial blood. Hence paroxysmal cough, of whatever nature, but especially that of pertussis, may lead to a general convulsion. Possibly, in whooping-cough, convulsions may be in part due to the spread, in the predisposed brain, of the "discharge" causing the cough. It is doubtful whether convulsions ever result from active congestion of the brain, which was once thought to be their common cause. If the condition ever has this effect, it is only in excessively rare cases, or when the congestion is the first stage of inflammation. During the first few days of life convulsions are not uncommon, but they probably always depend on direct injury to the brain during the process of birth (see p. 414). At all periods of infancy and childhood they may be due to the occurrence of an acute cerebral lesion, but these cases do not come within the class considered in this chapter.

Symptoms.—Infantile convulsions are often preceded by other signs of disturbance of the nervous system. The child is restless and irritable; the aspect of the face often changes; there may be a little twitching of the mouth at times, or some abnormal movement of the eyes. In rickets, there is often inversion of the thumbs and great toes—"carpo-pedal contractions"—or there have been attacks of "laryngismus stridulus," or more distinct partial convulsions. The actual attack usually comes on suddenly, and may occur during either the waking or the sleeping state. Sometimes a severe attack resembles closely an epileptic fit, but in general the attacks are slighter than typical epileptic convulsions. There is a sudden fixation of the eyes, which are often rolled up or down, or to one side, or may converge or diverge. The face and lips become pale. The limbs and trunk are rigid and stiff, the head turned to one side or retracted. Respiration is interfered with, and the face becomes dusky. After a minute or so, the spasm may relax, or (less frequently) may become clonic as in an epileptic fit. Sometimes the spasm is entirely clonic, but it is then slight, and involves chiefly the face and eyes, or the hands and feet. In other cases, again, a slighter tonic spasm may continue for a considerable time, half an hour or more; it is then insufficient to stop breathing, but may interfere with this enough to cause a slight change in the colour of the face. In these cases the spasm may not change the position of the limbs, and may not be noticed until the alteration in the colour of the face attracts attention, when the rigidity is discovered. Such persistent spasm, however, usually varies from time to time, so that there is really a series of convulsions, with imperfect recovery in the intervals. Many slight attacks are accompanied by a scream, and are popularly called "screaming fits;" there may be nothing more than a scream and quick breathing or grinding of the teeth, and brief rigidity of the limbs. Such convulsions are probably always bilateral; it is doubtful whether the fits in true infantile eclampsia are ever confined to one side.

The attacks termed "laryngismus stridulus" consist of laryngeal spasm, analogous to the convulsions, and under the same conditions. They consist of sudden spasm, stopping the breath and causing the child to seem on the point of death from suffocation. Then the spasm relaxes, and, with a loud crowing inspiration, as the vocal cords recede, the child gets its breath again. Such attacks often occur many times daily, spontaneously, or excited by some sudden alarm, or by some peripheral impression.

The convulsions of rickets may be few, and cease after a few days, or they may recur during several weeks, or even months. Not uncommonly they begin at eight or nine months, and go on through the second year of life, and then cease. They may, however, continue for a still longer time, and the condition must then be regarded as epilepsy. Indeed, whenever attacks continue after their cause has ceased, the condition is inseparable from epilepsy. Some of the children in whom the fits cease after lasting for a year or so, become epileptic in later childhood or at puberty.

Diagnosis.—The chief point in the diagnosis of the nature of infantile eclampsia is to distinguish from it the convulsions that are due to organic brain disease. Those produced by such processes as tumour or meningitis are soon accompanied by other symptoms of the morbid process. It is important, however, to bear in mind the fact that a commencing organic process may increase the irritability of the brain before it causes other symptoms, and then some peripheral impression may excite a convulsion. It must be remembered that a fit, even in a young child, generally means some increased central excitability, and the older the child the greater is its significance in this respect. If there is not evidence of a cause of increased excitability (such as prostration or rickets), the possibility that there may be commencing organic disease should always be remembered.

A special difficulty is presented by the cases in which convulsions are due to a sudden cortical lesion and recur (see p. 750). The distinction of these fits from those of eclampsia rests especially on the fact that they are generally unilateral, and often commence locally in some part of one side. When they are accompanied by distinct evidence of hemiplegia, corresponding in side to the convulsions, the diagnosis is easy. Often, however, the paralysis is slight and unnoticed. Whenever fits are one-sided, and commence with a sudden series of severe convulsions, accompanied by the signs of a cerebral illness, it is exceedingly probable that they are of this nature. If recurring eclamptic convulsions are ever unilateral, they are not constant in seat, but affect, at different times, one side and the other.

In all cases of infantile convulsions a careful search should be made for any peripheral cause of irritation, such as local suppuration, intestinal worms, and the like.

PROGNOSIS.—There is more danger to life in the eclamptic convulsions of infancy than in the epileptic fits of later life. Frequent convulsions may readily cause fatal exhaustion in a young and feeble child. The actual prognosis must be founded on the severity and frequency of the fits, and on the strength of the patient. If convulsions continue for some months, the prognostic question arises—Will the disease go on to epilepsy? The danger is considerable, and it increases the longer the fits continue. But even after they have lasted for a year, there is a fair chance of arrest.

TREATMENT.—In the treatment of the convulsions of children, the first important measure is to search for their cause, and, if possible, to remove it by treatment. This is especially important in the case of rickets, in which the mere treatment of the attacks may have little influence if their cause is allowed to continue unchecked, whereas the treatment of the underlying diathetic state, by cod-liver oil and steel wine, may alone speedily stop the fits. The attacks themselves, whether general convulsions or the local laryngeal spasm, are generally amenable to bromide of potassium, but it is necessary to give this in adequate doses, three grains to a child under six months; five grains to one between six and sixteen months, and still larger doses to older children. Two or three doses may be given in the day. Severe attacks may need the inhalation of chloroform, which is almost always effective, but may have to be repeated two or three times before the bromide that is given can assert its influence.

It was formerly the custom to treat infantile eclampsia by warm baths and by applications designed to draw the blood to the skin, on the theory that the convulsions were the result of cerebral congestion. Grey powder was given, in frequent doses, to combat the supposed local condition. Indeed, the fact of a patient having suffered from infantile convulsions may still often be correctly surmised from the indications presented by the permanent teeth of the influence of the mercury in early life. The theory was certainly erroneous, and it is doubtful whether the treatment did any good. I remember, when a pupil in the country, spending the greater part of a day watching in vain for the expected effect of repeated warm baths on the almost continuous tonic convulsion from which a rickety child was suffering. Certainly, unless a warm bath has an immediate effect, it is useless to repeat it.

The dentition theory of the origin of the convulsions led to the almost universal adoption of the practice of lancing the gum over a coming tooth to lessen the irritation. As the sole or even the chief element in the treatment, the measure is a mistake, since it deals with the least important, and often quite unimportant, element in the causation of the convulsions. It is, however, probable that the measure does sometimes facilitate the eruption of the tooth, and may thus lessen any irritation that exists.

PUERPERAL CONVULSIONS: PUERPERAL ECLAMPSIA.

ETIOLOGY.—Towards the end of pregnancy, during labour, and after labour, women sometimes suffer from severe convulsions, and of those thus attacked, many die—from 20 to 30 per cent. Women who are pregnant for the first time, and those who are comparatively young, are the most prone to suffer, and in them the malady is most fatal. A few of the patients have been epileptic, and the fits are simply those to which they have long been liable. In most cases of epilepsy, however, the attacks do not occur during the puerperal period. In some other cases, very rare, there has been evidence of no other causal condition than an over-excitable state of the nervous system, and the convulsions were distinctly excited by the pains of labour, or by the irritation of digital examinations. In the vast majority of cases, however, puerperal convulsions are associated with the presence of a large quantity of albumen in the urine, in which casts also are found, and urea is deficient in quantity. In fatal cases, the kidneys present indications of prolonged congestion, and often a condition that has been generally regarded as one of nephritis. These signs of kidney disease may be accompanied by distinct œdema of the subcutaneous tissue. The association of puerperal convulsions with such evidence of altered function of the kidneys is too frequent to be without very strong significance, and the balance of evidence is strongly in favour of the common inference that has been drawn from the association—that puerperal convulsions are really uræmic convulsions, or at least due to the retention in the blood of material that should be eliminated by the kidneys. They differ from such convulsions under other circumstances in the common absence of pronounced uræmic coma, but they differ also in another fact, which explains to some extent the absence of coma—the presence of a powerful cause of reflex irritation, which, as we have just seen, may excite convulsions apart from any toxæmic influence, and when no other predisposition exists than an undue central excitability of the nervous system. We must remember, also, that the condition of pregnancy itself involves an abnormal state of the nerve-centres, as is clearly shown by the occurrence of many functional disorders, such as chorea and tetany. Thus the influence of the blood-state is exerted on predisposed centres, and is aided by peripheral irritation, which is, under the circumstances, peculiarly powerful. Moreover, even when other signs of uræmia are absent, albuminuric retinitis often testifies to the intensity of the influence on the system exerted by the renal disease, and is a proof of the essential correspondence of the state with that of Bright's disease, which has been overlooked by many writers.

The evidence is so conclusive that it seems hardly worth while to mention the objections that have been raised to the theory, or the hypotheses that have been put forward to take its place. In addition to the absence of other signs of uræmia, which has been already discussed, it has been alleged that albumen is to be found in so large a proportion of pregnant and puerperal women that its coincidence with convulsions has no significance. But it is a question not of the mere presence of albumen, but of its amount, and the amount that is common apart from convulsions is trifling and without significance.* On the other hand, clinical experience shows that whenever a woman, at or near labour, presents much albumen in the urine, with other signs of nephritis, she is in imminent danger of convulsions. The chief other theory that has been put forward is, that there is anæmia of the brain, the result of a watery state of the blood and of vaso-motor spasm, which is itself due to the influence of irritation of the uterine nerves or of the nerves of the sacral plexus. To render this theory tenable it has yet to be proved, first, that there is more vaso-motor spasm than frequently exists in various conditions without convulsions; and secondly, that vaso-motor spasm ever yet gave rise to a fit. The only hypothesis that deserves future attention is that the renal condition, and perhaps the convulsions also, are due to microbial infection of the blood. This has been supposed to be due to infarction of the placenta, in which white nodules have been found, containing bacilli that are said to cause convulsions in pregnant animals. But more facts are needed to put the theory on a current footing.

SYMPTOMS.—Puerperal convulsions occur during, before, or after labour. They may not only be distinctly excited by the local irritation, but are sometimes set up by other causes, as a blow on the head (Engstrom), while the depression of anxiety and fear seems sometimes to aid in their production. In addition to the œdema already mentioned, their onset is often heralded by severe headache, precordial pain, or sudden amaurosis, the latter certainly and the others probably uræmic in origin. The onset is usually sudden, and the convulsions generally resemble closely those of epilepsy. There is absolute unconsciousness, the tongue is frequently bitten, and the bladder and rectum may be emptied during the fit. One difference from ordinary epileptic fits is, that very frequently the affection of the two sides is not simultaneous, but successive. The eyes are often conspicuously involved. In one case in which the character of the fits was very carefully noted* each began with nystagmus, lateral and to the left. Then first the eyes and afterwards the head were strongly turned to the left, and clonic spasm came on in both eyelids, with tonic contraction of the left side of the face, and the left arm and leg. First the wrist was extended, then strongly flexed and pronated. Subsequently the eyes and head turned to the right, and the right arm and leg were rigid like the left, so that tonic spasm became universal, and the tongue was thrust out of the mouth; then general clonic spasm set in, with horizontal nystagmic movement of the eyes. The spasm ceased on the left side sooner than on the right. During the coma that followed, the eyes were rolled upwards. All the fits were the same, save that in the earlier attacks there was opisthotonos, and the later fits, as the patient became exhausted, were limited to the face, involving especially the frontales. The patient was a young married woman aged eighteen, who had an intense dread of parturition. The first fit commenced soon after labour set in, and each pain excited a convulsion. She was delivered with the forceps, but the fits continued, with an interval after labour of four hours, and she died forty-eight hours from the onset. The temperature rose to 103°, and continued at that point. The urine, when heated, became solid from albumen, and the kidneys after death presented the characteristic signs, naked-eye and microscopic, of acute inflammation. In some cases, in which the convulsions are very severe, the temperature rises to 108° or 109°, as it may do in the *status epilepticus*. The average mortality in puerperal convulsions is about 30 per cent.†

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* This point has been ably and conclusively discussed by Galabin, 'British Med. Journ.,' Aug. 30th, 1880.

TREATMENT.—The treatment is in part obstetrical, the most important element being the termination of pregnancy; and the problem of waiting for the child to be capable of living, and at the same time avoiding undue risk to the life of the mother, is one of the most delicate and difficult tasks in obstetric practice. For the convulsions themselves, venesection was formerly employed, and when much blood had flowed, the fits usually ceased; but the consequent exhaustion of the patient, and the discovery of the coincident kidney disease, have led to the abandonment of this practice, especially in view of the fact that inhalations of chloroform are usually successful in arresting each convulsion. As long as the inhalation is kept up, the convulsions may cease, but when it is discontinued they too often return. The treatment of the blood-state is necessarily limited under the circumstances of parturition, and yet it is unquestionably most important. Vapour-baths have been employed, and so have pilocarpine and nitrite of amyl, sometimes with success. Theoretically, venesection and transfusion might be expected to afford more chance of recovery to a desperate case than any other measure. For further details of treatment the reader is referred to works on obstetric medicine.*

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* By Dr. Hyde Marriott, formerly of University College Hospital, to whom I am indebted for the report of the case.

† Of 62 cases in the Königsberg Hospital Records, the percentage mortality of mothers was 29, and of children 38 per cent. (Gettkant, 'Diss. Königsb.,' 1881).

Uræmic Convulsions.

The nature and precise cause of the convulsions of uræmia are topics beyond the province of this work, but it may be convenient briefly to describe their characters. They occur only when the kidney disease has profoundly affected the system and has altered the state of the blood; in chronic kidney disease there are usually other indications of this effect in hypertrophy of the heart and albuminuric retinitis.

Uræmic convulsions usually set in suddenly, and there may be no preceding symptoms to indicate what is coming. In some cases, however, they are preceded by other symptoms of uræmia, as coma or amaurosis. The fits are seldom isolated; usually several occur in a short time, sometimes in the course of an hour, sometimes at intervals of several hours.

The convulsion usually resembles very closely an epileptic fit, consisting of tonic and clonic spasm. The preponderance of the spasm on one side is generally well marked, and causes a strong deviation of the head and eyes to one side. Occasionally the convulsion is unilateral, but the affection of one side is not constant,—the side involved varies in different fits. Another occasional characteristic is a special affection of the muscles of the face, which may be bilateral, as was conspicuous in the case of puerperal convulsions described above. Nystagmus, as in that case, is sometimes observed, the quick movement being to the side towards which the head deviates. The convulsions often begin locally, now in one part, now in another, but quickly spread. The pupils are usually dilated during the fit, and do not act to light, but I have known them to be contracted, and to dilate afterwards. In severe convulsions the interference with respiration is very marked, and the tongue may be bitten. Immediately after the fit, the knee-jerk may not be obtained. The patient may sleep for a time after the attack, as after an epileptic fit, or may pass into a state of coma, or may be merely drowsy.

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* Many papers relating to this affection, by Galabin, Banner, and others, will be found in the 'Brit. Med. Journ.,' 1891, vol. ii.

The treatment of uræmic convulsions is that of the blood-state.

VERTIGO.

The term "vertigo" means, by derivation, a "turning," and is used as a designation for any movement or sense of movement, either in the individual himself or in external objects, that involves a defect, real or seeming, in the equilibrium of the body. The word "giddiness" is used as a popular synonym for vertigo. An exact and complete appreciation of the relation of the body to its physical surroundings is involved in the mental state that we call "consciousness." If that appreciation is incorrect in any degree—and it is rendered incorrect by a false sense of movement—to that degree the sense of perfect consciousness is interfered with. Hence vertigo always involves a slight interference with consciousness. This fact influences the popular use of the term "giddiness," which is sometimes used to designate a mental state of imperfect consciousness when there is no sense of movement, and in which there is nothing that would be termed "vertigo" in the strict medical use of the word. The word "dizziness" is more often used in the sense of imperfect consciousness, but it is also sometimes used as a loose synonym for "giddiness."*

Vertigo is a common symptom of organic brain disease, but in such cases it is associated with other distinct symptoms of its cause, and has been already considered (p. 97). In all cases, indeed, it is merely a symptom of some definite morbid state, but in many conditions it is a symptom that is far more obtrusive than its cause. Hence it has become customary to describe vertigo as if it were a definite disease, and the method has considerable practical convenience, although it is not logically correct.

Before describing the special forms presented by vertigo, it is desirable to consider what is known of the symptom and its origin. The subject involves a very obscure branch of the pathological physiology of the nervous system. Vertigo is essentially a sense of involuntary movement, but it may be perceived only as an apparent movement of external objects,—"subjective," and "objective" vertigo. It involves a sense of defective equilibrium, and it is necessary to consider, first, what we know of the way in which equilibrium is maintained under normal circumstances.

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* The original meaning of "giddy" was mirthful (a sense still extant), and that of "dizzy" was foolish or dull.

The maintenance of equilibrium is effected, except when the body is at rest, by the activity of the muscles, influenced by the brain. When the body is at rest, the equilibrium is permitted by the absence of muscular contraction. The action of the brain is in some way determined by certain centripetal impressions, which give information to the cerebral centres as to the relation of the body to external objects, as to the manner in which the body is supported, and as to its position. (1) Sensory impressions come from the skin, &c., of the parts on which the body is resting. (2) Centripetal impressions reach the brain (probably in part through the cerebellum) from these muscles, the contraction of which determines the posture of the body and its support; the most important of these are from the muscles of the legs and lower part of the trunk. (3) Information as to the position of the eyes and head shows the relation of seen objects to the body; this information is chiefly derived from the sense of the active innervation of the muscles (see p. 173), probably aided by centripetal impressions from them. (4) Impressions from the semicircular canals of the internal ear, which are apparently due to the pressure of the endolymph, under the influence of gravitation and of movement (Flourens, Goltz, Cyon,* &c.). We shall have to consider these in greater detail presently.

Of these various impressions, which are doubtless always acting on the intra-cranial centres, only those of the first class are felt as sensations; the others seem to give rise, in some way, to perceptions—of posture, &c.,—which are not true sensations. They apparently act on some centre through which equilibrium is maintained, and which in some way regulates the outgoing currents of nerve-force, probably the middle lobe of the cerebellum. We can trace a connection between it and many of the afferent paths concerned. It may merely co-ordinate these impressions, and influence, according to them, the cerebral centres whence the outgoing currents proceed, and the states of these motor cerebral centres, thus induced, may influence consciousness as the "perceptions" alluded to.† This idea harmonises the various facts better than the assumption that the outgoing impulses proceed directly downwards from the cerebellar centre, of which there is no evidence, direct or indirect.

If any one of the centripetal impressions that regulate equilibrial co-ordination is imperfect, we are chiefly conscious of the effect on the action of the centre, not of the imperfection itself. It is thus that

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* The various experiments on the subject are described in most text-books of physiology. Their details, however interesting, have not such a direct bearing on the medical aspect of vertigo to make it worth while to describe them here.

† What may be termed the perceptive character of the influence on consciousness —its origin in processes not felt directly—is illustrated by the fact that there is sometimes a feeling that objects are moving, combined with a consciousness that they are not. Some patients describe a feeling of impending giddiness as being as distressing as actual giddiness.

"vertigo" seems to arise, at least in many conditions. That vertigo is commonly a disturbance of the action of some centre, and is not a mere consciousness of the defective centripetal impression, is shown by the fact that, when well marked, it is clearly a sensation of a motor process.* There may be merely a sense of movement, but if the sensation is intense, there is actual movement, and this is in the same direction as the sensation of movement.† From this it appears that the sensation felt is the result of the process which, in greater degree, causes the movement, *i. e.* it is due to a motor process. Even when the apparent movement is in external objects, the same correspondence is often observed; if the patient moves, or seems to move, it is often in the same direction as that in which objects appear to move. This is intelligible only on the assumption that the sensation is due to a motor process. If a person actually moves, and an object remains opposite his face (in the centre of his field of vision), he rightly infers that it has moved. The subjective process in vertigo leads to the same inference. Sometimes, however, objects appear to move in the opposite direction to that of the sense of subjective movement; to this we shall presently return.

These statements apply to what may be termed typical vertigo from derangement of guiding impulses. But vertigo may be due to other causes. We have seen that it is common in epilepsy as the warning of severe, and the sensation in slight, attacks. We can understand its occurrence in these cases on the theory just described. In epilepsy there is a spontaneous motor discharge, which may be one-sided, or merely greater on one side than on the other; in either case there will be a tendency to lateral movement, which is actually seen in the deviation of the head and eyes, and may give rise to positive rotation. This motor process may be felt as vertigo before it causes movement, or when it is too slight to cause movement. Hence, too, we can understand that many organic lesions of the brain should cause vertigo at the time of their occurrence, just as they may also cause convulsion.

But the sensation is not always thus definite, and does not always correspond to the motor process. It may be merely a vague sense of unsteadiness or uncertainty. It is possible that in some cases it may be a more direct effect of the deranged and discordant centripetal processes, or the vagueness of the sensation may be due to the trifling character of the motor disturbance, or elements in its production which we cannot yet clearly discern.

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* This fact has been repeatedly insisted on, as of paramount importance, by Dr. Hughlings Jackson. It was also pointed out, long ago, by Dr. Russell Reynolds: "The feeling of equilibrium results from the harmony of our different sensations among themselves, and with the motor impulse which is their combined effect. When any one group of the sensorial impressions is distorted or removed, the balance is disturbed, and as these impressions are themselves the stimuli of muscular action, attempts are made for its restoration, ... producing vertiginous or allied movements."—Russell Reynolds, 'Vertigo,' London, Churchill, 1854,—a paper in which are collected all the older facts and opinions on the subject.

† It may be partial, as in a patient in whom a tendency, in walking, to deviate to the left was accompanied by an irresistible impulse to put the right leg across the left, which often caused a fall.

The mechanism of vertigo is thus so complex, and our knowledge of it is so fragmentary, that we may easily go very far wrong if we endeavour to formulate the precise mode in which any given sensation is produced, or the actual disturbance that exists in any given case. But at the same time we may note the apparent suggestiveness of some of the phenomena, in considering their features. The exact character of the sensation experienced is often very difficult to ascertain; and this is not surprising, for the movement is one of intense mental confusion, in which accurate observation is most difficult.

The apparent movement of the individual himself, the subjective vertigo, as we have seen, varies much in different cases. When it is definite in character, its direction is often emphasised by the fact that the patient falls or tends to fall in the same direction. There may be a tendency to incline to the right or left, or to turn towards right or left, but more often this is combined with a movement forwards or backwards. Sometimes there is a sense of going downwards—of sinking. One patient described it as if there were a powerful magnet in the earth, drawing him towards its centre; another, as if there were a huge weight on his head, pressing him downwards. A sense of falling from a height is also common, and is probably familiar to most persons as a sleep-sensation, the cause of which will be considered presently. It is noteworthy that the sense of going downwards towards the earth may have the same character, whether the patient is standing or lying. This sensation is occasionally felt when there is disease of the semicircular canals, and it appears, therefore, as if the precise direction of the false sensation were determined by the true sensations engendered by the posture of the body. The patient who feels as if he were sinking into the earth when he is standing, feels as if he were sinking through the bed when he is lying. The opposite sensation, that of rising, is uncommon, but one form has been described to me as a sense of "walking on the air," or as if, when sitting, the body scarcely touched the chair.

The apparent movement of objects, objective vertigo, may be an impression that they are passing before the patient in a given manner, lateral, upwards, or downwards, as we have seen, which usually corresponds to that in which the patient seems to move. It is usually combined with subjective vertigo, and intensifies the distress, so that the eyes have to be kept closed in severe cases.* In some instances, however, the direction in which objects seem to move is opposite to that of the subjective movement. The explanation of this is difficult, and may not be always the same. In some cases the disturbance may not involve the visual centres in such a way as to give rise to the error of inference, which causes the apparent movement of objects in the same direction as the subjective movement. If a person in health turns round, objects seem to move in the opposite direction, because their images move across the retina; in some cases of vertigo there may be a false impression of such a reverse movement, although there is no movement of the images on the retina.* In the same way, to a patient who fell forward, it seemed that the ground rose up to meet him, while to one who, in bed, felt as if rolling over to the left, the left side of the bed on which he was lying seemed to be rising up. This condition, in which objects seem to move, as they would seem if the person moved but the objects did not, is perhaps a less complete form of vertigo than that in which objects seem to move *with* the individual.

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* Even opening the eyes in the dark, when no object can be seen, may intensify the vertigo, an illustration of the closeness and extent of the association of the use of the eyes and the maintenance of equilibrium.

In other cases again, objects before the eyes seem to move in a manner that has no exact parallel in the subjective vertigo. Every object may seem to be rotating on its own axis, or all the objects may seem to rotate around a fixed point; rarely objects rotate towards the patient. In these cases the subjective movement may be a simple one to right or left, or there may be no subjective vertigo; the apparent rotation of external objects may be the only sensation of which the patient is conscious. The disturbance, in such cases, is probably complex, and we cannot yet discern its possible mechanism.

There seems to be a special relation between the labyrinthine impressions and the movements of the eye, and in labyrinthine vertigo there may be an actual involuntary movement of the eyes. This is probably the slightest degree of the actual movement which, as we have seen, occurs in many cases, and involves the whole body; whenever there is actual rotation, the eyes and head lead the way.

There is a close association of vertigo with vomiting, and often also with disturbance of the vaso-motor system; in vomiting from any cause there is often a "cold perspiration." Intense giddiness almost always causes vomiting. It is probable that the gastric fibres of the vagus have a connection with the middle lobe of the cerebellum, disease of which so frequently causes vomiting. It is more probable that the symptom is thus produced than that it is due to a direct connection between the auditory and vagal nuclei. It has also an obverse side in the influence of gastric derangement in exciting vertigo, which we shall consider presently.†

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* Objects are not then *involved* in the false inference, as in the vertigo described as typical, but there is, as it were, a subsidiary false inference of their movement in the opposite direction. Another possible explanation is that the apparent movement of objects is the result of compensatory processes to maintain the disturbed equilibrium.

† It probably underlies the pathology of sea-sickness, in which the vomiting may be simply a reflex effect of the unaccustomed oscillations of the endolymph in the semicircular canals.

OCULAR VERTIGO.

Ocular vertigo is a rare form; it is due to weakness of an ocular muscle, and depends on the erroneous projection of the visual field (see p. 172). There is an error in the unconscious inference of the relation of the body to seen objects, and this element in the impressions that influence the equilibrial centre is at variance with others, and either the discord or the disturbance of the cerebral motor centres induces giddiness. The sensation is seldom intense, and is not independently paroxysmal; it occurs only when the affected muscle is put in action. In most cases it is slight and transient, as the cerebral centres learn to neglect the error. Rarely it continues for a long time. It is chiefly important in respect to diagnosis. The treatment of the ocular palsy soon removes the vertigo, but if its cause is not discovered, the patient may be put through long courses of useless treatment.

AURAL VERTIGO; LABYRINTHINE VERTIGO; MENIÈRE'S DISEASE.

The vertigo that depends on disease of the semicircular canals is sometimes termed "aural" or "auditory vertigo," sometimes "labyrinthine vertigo," sometimes "Menière's disease." It always depends on derangement of the auditory nerve, commonly on disease of the nerve-endings in the labyrinth (or of their disturbance by disease of the middle ear), rarely on disease of the fibres of the nerve in its trunk or origin. Attention was first drawn to this cause of vertigo by a description given by Menière* of some cases in which very violent disturbance was produced by a sudden lesion of the labyrinth. Such cases came to be termed "Menière's disease"—the great frequency with which slight disease causes slighter symptoms was not at first recognised. This name has been since applied, by some, to all forms, slight and severe; by others it has been more wisely restricted to the severer cases.

This labyrinthine form is by far the commonest of all varieties of vertigo. In nine cases out of ten in which there is definite giddiness, not epileptic in nature or obviously due to organic brain disease, it is due to a morbid state of the labyrinth or auditory nerve-endings. It has been doubted by some whether this opinion is justified, whether undue significance is not given to the association of slight deafness with the vertigo; but the evidence, considered as a whole, is very strong. In the first place, we have the undoubted fact that violent vertigo may result from acute lesions of the labyrinth. Secondly, progressive dis-

* Before the Académie de Méd. of Paris, in 1861. The association of tinnitus aurium and vertigo was noted by Burns in 1809 ('Obs. on Dis. of the Heart,' p. 75).

ease of the labyrinth may be attended with severe vertigo, which may cease when the progress of the disease has produced complete deafness. These two facts suggest that the vertigo is the result rather of the irritation of the nerve than of its loss of function. Thirdly, in the majority of cases of definite vertigo, slight or considerable, tinnitus is present, and is evidence of a process of irritation of the fibres of the part of the nerve concerned in hearing. Fourthly, slight loss of hearing, tinnitus, and vertigo may be observed not only to come on together, but to pass away together. Lastly, the frequency with which symptoms of labyrinthine disease are found in cases of vertigo is itself a fact of very great significance. Of 106 consecutive cases in which definite vertigo made the patient seek advice, in no less than ninety-four, ear symptoms were present, tinnitus or deafness, or more often both; defect of hearing through the bone always existed, and in almost all cases in which it was slight, a distinct difference between the two sides emphasised its pathological character. In conjunction with these facts we have the experimental evidence that vertiginous movements result in animals from lesions of the semicircular canals.*

The coincidence of auditory symptoms with vertigo depends on the fact that the lining membrane of all parts of the labyrinth is continuous, and so also is its cavity. The two sets of symptoms depend on the affection of adjacent structures, which commonly suffer together; but since they are distinct, it is conceivable that labyrinthine vertigo may occur without any auditory symptoms. I have seen two or three cases in which definite giddiness existed alone, and in which auditory symptoms came on later, and many in which the defect was limited to inability (on one side only) to hear the high-pitched notes of Galton's whistle, or to perceive the sound of a watch through the bone. From this consideration it is easy to understand that there is no necessary proportion between the disturbance of hearing and of equilibrium.

The central relations of the two parts of the auditory nerves have been already described (p. 47). The stimulation of the nerve-fibres of the semicircular canals is believed to be through the pressure of the endolymph on the hairs within the ampullæ acting on the epithelioid elements in which the nerve-fibres end. The three canals are arranged at right angles; the horizontal and transverse canals have their ampullæ at the outer ends, while that on the vertical canal is at its antero-inferior extremity. If movements stimulate the nerves by increasing the pressure in the ampullæ, the fibres of the transverse canal will be excited by an inclination of the head to the opposite side, as in falling in that direction; those of the horizontal canal by a rotation of the head towards the side of the canal concerned; those of the vertical canal by a forward movement. Irritation of the canals by disease may be expected to give rise to corresponding sensations of movement. But it is probable that, in most cases, the condition is a complex one, and the disturbance is seldom the result of the irritation of a single canal.

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* The literature of aural vertigo is exceedingly voluminous. Among the more important papers are those of Knapp and Brunner, 'Arch. of Oph. and Otol.,' vol. ii; Hughlings Jackson, 'Med. Times and Gaz.,' 1872, 'Med. Record,' vol. ii, and 'Lancet,' 1880, Oct. 2nd; Charcot, 'Prog. méd.,' 1874; Buzzard, 'Lancet,' 1890.

On account of the frequency with which vertigo seems to the patient himself to preponderate over the auditory symptoms, a large proportion of the sufferers seek the advice of physicians rather than of aural surgeons, and most text-books of diseases of the ear give a very imperfect representation of the malady. The following account is based upon about 150 cases in which the affection was distinct.

ETIOLOGY.—Aural vertigo seems to result from almost any of the many morbid processes that involve the labyrinth and the nerve-endings it contains. As a chronic symptom, however, it seldom results from inflammation of the middle ear or caries, such as are so common in childhood. It is a well-known fact that in these cases the labyrinth usually escapes.* In most cases there are no signs of mischief in the middle ear; the affection seems to be confined to the labyrinth. Chronic slightly irritative disease is that which seems most frequently to cause it, and such disease is most common in the second half of adult life. It is met with very seldom under twenty, occasionally between twenty and thirty, and frequently after thirty. In four fifths of the cases the symptoms commence between thirty and sixty. For some reason, it is more common in men than in women, in the proportion of about two to one.

The nature of the change in the labyrinth is necessarily a matter of conjecture. In a few cases the symptoms came on after exposure to cold, and inflammation is the probable lesion. Such inflammation is generally chronic or subacute, but in one of Menière's cases, due to cold, it was so intensely acute that the patient died in a few days from the severity of the cerebral derangement produced. In many cases the mischief seems to be the result of gout, acquired or inherited, and it is highly probable that the labyrinthine membrane may suffer from this cause, as other membranes do that are connected with bones. I have more than once known all symptoms to pass away under the influence of treatment directed to the gouty state. Occasionally a syphilitic inflammation is suggested by the presence of the constitutional disease, and labyrinthine lesions have been proved to occur from this cause.† In other cases, again, the slow progressive character of the symptoms suggests that they are due to degenerative changes in the membrane, either senile in character or allied to senile changes. Atrophy of the nerve may be a cause, but exists more rarely than symptoms suggest (see p. 262). It is sometimes met with, in characteristic form, in tabes. Very acute inflammation or hæmorrhage, such as gave rise to the intense symptoms of the cases described by Menière, is certainly most rare. Another very rare cause is a lesion at the base of the brain, damaging both the auditory and facial nerves, but such cases do not come into the class now under consideration, in which the vertigo itself is the chief symptom. It should be noted that attacks may be excited by almost any depressing influence in a predisposed subject. The effect of stomach derangement will be again considered, and, in addition to this, I have known attacks to be brought on by fatigue, by excitement of various kinds, and by pain, such as toothache.

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* In a curious case reported by MacBride the symptoms were caused by disease of the middle ear through the agency of defective pressure in the tympanum, and were at once relieved by inflation. (Quoted by Grainger Stewart, 'On Vertigo,' 1884, p. 20.)

† As in a case of acute deafness and giddiness in a syphilitic subject (recorded by Moos, 'Virchow's Archiv,' Bd. lxix), in which the whole labyrinth was occupied by semi-solid inflammatory material.

SYMPTOMS.—The vertigo in these cases presents very great variation in its character. It is almost always paroxysmal, but there is often continuous slighter vertigo, sometimes vague, often of the same character as that in the attacks. Less commonly there is no vertigo except during the paroxysms, or for a few hours or a few days afterwards. The attacks may occur at intervals of a few days or of several weeks or months; sometimes many attacks occur daily. They may come on spontaneously, or be excited by some sudden movement, occasionally by coughing or sneezing. Some of these patients can never blow the nose without reeling. An attack may come on during sleep, and wake the patient.

The vertigo may be subjective or objective, or both. When the patient feels as if turning or falling, there is often an actual tendency to turn or fall in the same direction during the severe attacks. A tendency to go forwards or backwards is very common, with or without a lateral tendency. A sense of movement in external objects is also frequent, and this may present all the varieties described at p. 780. When both phenomena are combined, it is far more common for the two movements to correspond in their direction than for them to be in opposite directions. The other aural symptoms are often one-sided, or are much greater on one side than on the other; in such cases the sense of movement may be either towards or from the ear most affected, but when the subjective and objective movements correspond in direction, they are more often towards the affected ear than from it. In one case of actual movement from the affected side, there was a sense of compulsion which could not be resisted. When they differ, it is about equally common for the subjective movement to be either towards or from the ear affected, and this is also true of the apparent movement of objects. It must be remembered that when there is not much difference in the auditory symptoms on the two sides, the excess does not furnish any strong ground for inferring that the cause of the vertigo is greater on that side, because the derangement of the canals may be greater on the side on which the cochlea is less affected.

The paroxysms of vertigo are generally sufficiently severe to make it difficult or impossible for the patient to stand, and in many instances he occasionally falls. Sometimes, indeed, the sufferer is hurled to the ground with violence, as if by some unseen power, much to his own astonishment and alarm. In one woman, the attacks began with a sudden irresistible impulse to run forwards a few yards and then to turn to the right, which made her fall to the ground, and for two hours there was a sense of movements of herself and objects to the right (the deaf side). Occasionally there is a sensation as of a blow behind the ear. A patient who felt as if rapidly whirled round from the deaf side had also a sense of pressure on the side of the head, seeming to make her turn. When the sufferer falls, it may be impossible for him to rise, so intense is the giddiness. In some very severe attacks there is a moment's loss of consciousness, as might be expected from the intense derangement of the functions of the brain. This occurs chiefly in attacks that are very sudden in onset, and in which the subjective giddiness is very intense. There is no succeeding "dazed" feeling as in epilepsy, but the vertigo continues, often for hours. In other cases, sight may be indistinct for a few minutes. After severe vertigo has lasted for a short time, nausea comes on, followed by vomiting; the patient becomes pale, and a cold sweat breaks out. The pallor and physical depression are often extreme, and very alarming in aspect. If the giddiness persists, the vomiting may go on for some hours, and after the stomach has been emptied, bile is brought up, as is usual in continued vomiting; this is popularly regarded as conclusive proof that the attack was due to "biliousness." The vertigo is often increased by any movement of the head, and the patient may be unable to raise the head from the pillow without being sick. Gradually, however, the attack passes away, but for a few days the patient experiences more than the ordinary amount of vertigo, and the interference with the functions of the stomach occasions some indigestion, and especially some diarrhœa. Vaso-motor disturbance sometimes precedes the onset, and connected with it are various cephalic sensations, from general pulsation, greatest in the head, or a "rushing to the head," to a sense of "something passing over the brain," and other allied sensations.

Often the paroxysms are much slighter in character, and are unattended by vomiting, or even by nausea. There may be merely a sudden tendency to fall, or sudden movement of objects, or a tendency to deviate to one side in walking. Attacks sometimes come on at night, and may be distinctly related to the horizontal posture, so that the patient has to sit up from time to time to get rid of the giddiness. This is probably related to an affection of a special part of the semicircular canals. Paroxysms may also occur during sleep, and wake the patient. The duration of the paroxysms varies from a few minutes to several hours, and bears a general correspondence to their severity. Severe attacks may occur at intervals of a few weeks or months, or even years. Between them there is often slight persistent unsteadiness, or occasional periods of slight vertigo, or the patient may be perfectly free from giddiness.

Ocular symptoms, secondary in origin, are present in some instances.[*] In cases of ear disease, an increase of pressure within the ear, as by pressing firmly the antitragus over the opening of the meatus, may cause nystagmus. During paroxysms of vertigo the patient may be conscious of a jerky movement of objects, a quick motion in one direction and slow return, like that sometimes produced by nystagmus, and I have known it to correspond with intermitting tinnitus. This apparent movement may sometimes alone be caused by pressure on the meatus;[†] and nystagmus may be produced, with vertigo, by disease of the middle ear, of course through the secondary affection of the labyrinth. It has been known to persist after the ear disease was cured, even for ten years.[‡] I have several times known double vision to occur during or after a paroxysm: in one case of pure aural vertigo, each attack was followed by double vision, jerky movement of objects, and distinct erroneous projection in the direction of the movement, so that, if the patient attempted to touch an object, the hand went too far in that direction. Slight diplopia is sometimes due to nystagmus that is not quite equal in the two eyes. It is apt to cause an error in diagnosis.

In most of the cases the auditory symptoms comprise both tinnitus and deafness. These correspond in side, except in rare cases in which one is bilateral. The deafness may present any degree of intensity, but is generally sufficient to be a source of trouble to the patient, and always involves hearing through the bone (see p. 263). In the cases in which the loss of hearing is slight, and unknown to the patient, it is generally one-sided; the watch can be heard loudly through the bone on one side, and not at all on the other, or the notes of Galton's whistle (very high-pitched sounds) are inaudible on one side, and the loss is emphasised by corresponding tinnitus. On the other hand, absolute deafness is scarcely ever met with, perhaps because when all hearing is lost vertigo usually ceases.

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[*] According to Cyon, irritation of each of the semicircular canals produces its own special effect on the position of the eyes.

[†] In 'Brain,' vol. iii, Dr. Hughlings Jackson describes a case of aural vertigo in which objects appeared to move from the affected ear in nystagmic jerks, and quotes a case of ear disease in which pressure caused objects to appear to move towards the affected ear. In several cases a similar spontaneous movement has been described to me, and cases are not at all rare in which it can be produced by pressure.

[‡] Urbantschitch, 1884. In this association it is instructive to note that in one case a pulsating tinnitus, without vertigo, was associated with jerking movements of the body and head, the result, as it seemed to the patient, of an irresistible impulse induced by the sound. It doubtless depended on the influence of conjoined disturbance of the semicircular canals on the motor centres, and presents, as it were, a general extension of the tendency to nystagmus.

Tinnitus is present, at some time, in the vast majority of the cases, and in almost all is persistent. It may present most of the variations described at p. 270. A continuous sound is the most common; pulsating sounds are occasionally described, but are less frequent than in the tinnitus that occurs without vertigo. The intensity of the sound is usually moderate, and sometimes slight, but it often becomes more intense at the onset of a paroxysm of vertigo, and may then become very loud. Thus one patient, who habitually heard a noise like a distant waterfall, said that with the vertigo it rapidly increased in loudness, and was like an express train coming past a station, and as it became loudest "it seemed to force on the giddiness." It is exceedingly rare for the noise to be confined to the paroxysms of vertigo. On the other hand, it is not at all uncommon for no increase of the tinnitus to take place on the occurrence of the giddiness, and paroxysms of tinnitus may not coincide with those of vertigo.

An attack of vertigo, however severe and however alarming it may be to the sufferer, is attended with little danger. Nevertheless it is possible that, in a patient with a feeble heart, the prostration may go on to fatal syncope. The chief danger, however, is in the cases, fortunately extremely rare, in which the labyrinthine lesion is intensely irritating, and the cerebral disturbance induced is so intense as to lead to fatal exhaustion.

Among the complications of the affection we can scarcely reckon the signs of labyrinthine disturbance, since these are really part of it. Many sufferers present varied indications of nerve weakness, partly secondary to the vertigo and to the tinnitus (*q. v.*) which so often attends it, partly associated, and disposing the centres to greater derangement under the influence of the peripheral disorder. Other neurotic affections sometimes co-exist; true epilepsy may accompany independent aural vertigo, and the affection is sometimes met with in the subjects of migraine. These diseases may also aid in intensifying the central disturbance. Disorders of the digestive system are common, and both excite, and result from, the attacks, while some patients present conspicuous evidence of gout.

The course of aural vertigo varies according to the nature of the morbid process. If this is steadily progressive, the symptoms may only cease when all hearing power is destroyed. Happily, however, these cases are rare; in the majority, either the changes in the labyrinth are not progressive, and the tendency to giddiness lessens in the course of time, or else the tendency can be kept down by treatment. I have known perfect recovery to occur in many cases in which the attacks of giddiness were most severe.

PATHOLOGY.—The chief facts regarding the pathology of labyrinthine vertigo have been already mentioned, but one or two other points remain for consideration. We must refer the vertigo to changes in the semicircular canals, but we must bear in mind the fact that it must always be perceived through the medium of cortical centres, influenced by the derangement of lower mechanisms. The readiness with which these are disturbed must influence the result. The precise mode in which the affected labyrinth acts on the centres is still unknown. It probably varies much, but is more often by the stimulation of the fibres, rather than by simple diminution of function. This conclusion is suggested by the results of experiments on animals, and is in harmony with the fact that there may be gradual loss of function, to judge by progressive deafness, without vertigo. Thus the morbid process is more nearly allied to the irritation which causes tinnitus than to that which causes deafness. Loss of function seems chiefly effective when on one side only, and vertigo from bilateral destruction of the labyrinth is commonly transient.

It is doubtful whether we can refer the paroxysmal character of the affection directly to the labyrinthine disease. The labyrinthine irritation probably brings the centre for equilibration (or the centre in which the centripetal influences are co-ordinated) into a state of instability, in which a sudden violent derangement may occur on some slight exciting influence, or even without any excitant that can be traced. This conclusion is indicated by the extreme intensity of many paroxysms, in which the sufferer is hurled to the ground with convulsive violence, without any coincident indication of special aural disturbance. In some cases a slight aural disturbance may perhaps excite the paroxysm, but even when a sudden loud subjective sound occurs as the vertigo comes on, we cannot take this as proof of a labyrinthine irritation of corresponding intensity, because it is possible, and even probable, that the sound associated with the vertigo may be of central origin, due to the extension of the central disturbance to the centres for hearing. Through these, of course, simple tinnitus is perceived, and their over-action must cause subjective sensations precisely like those produced in the labyrinth. The sound may thus be rather part of the attack than an indication of its cause. This view of the nature of the symptoms enables us to understand that severe paroxysms may occur when the labyrinthine change is apparently slight, and also the co-operation of stomach disturbance in exciting the paroxysms.

Diagnosis.—The diagnosis of labyrinthine vertigo depends on the coincidence of vertigo of definite character with indications of derangement of the functions of the labyrinth,—tinnitus, and deafness not due merely to impairment of the conduction through the external meatus or middle ear. The indications of this have been already described (p. 263). If the loss of hearing is trifling, its significance is greater if it is one-sided. It must be remembered that the fact at paroxysms are excited by stomach disturbance does not prove that the vertigo is simply gastric. A gentleman had always been liable to attacks of acute dyspepsia, but they were never attended with giddiness until he became deaf; afterwards, each dyspeptic attack was accompanied by severe vertigo. The diagnosis of aural vertigo is occasionally helped by the fact that giddiness may be brought on by a sudden movement of the head in one direction and not in another, or by suddenly increasing the pressure in one ear.

Cases of epilepsy, in which the aura is an auditory sensation accompanied by giddiness, may be mistaken for labyrinthine vertigo. The distinction depends on the fact that in the latter there is usually persistent tinnitus, impaired audition, and more or less constant slight vertigo, while loss of consciousness is extremely rare, and is confined to an occasional very violent paroxysm. Such a paroxysm is followed by vomiting and by prolonged giddiness. In epilepsy, loss of consciousness is the rule; there is no subsequent vertigo, but a slow return of normal consciousness, and indications of a convulsive seizure can usually be ascertained. It must be remembered that the two diseases sometimes co-exist.

It is a very common thing for aural vertigo to be mistaken for a slight attack of organic cerebral disease, congestion, or an actual vascular lesion. The error is the more easy because both classes of disease are common in the degenerative period of life; but it generally arises from ignorance of the occurrence and frequency of labyrinthine vertigo. It is a mistake the more easily made, because the prostration and pallor that are the consequence of the giddiness are very like those that result from a cerebral lesion. The diagnosis rests on the absence of other symptoms of such a lesion on the one hand, and on the presence of aural symptoms on the other; while in most cases the decision is much helped by the fact that the patient has had other attacks of simple giddiness, which are as significant if they have been slight as if they have been severe. In a cerebral attack, loss of consciousness is out of proportion to the vertigo, whereas in the other it is slight, often imperfect, and clearly subordinate to the intensity of the giddiness.

PROGNOSIS.—The prognosis is distinctly serious chiefly in cases of steadily progressive disease of the labyrinth, in which the symptoms may persist in spite of all treatment, until complete destruction of the nerve-endings brings a cessation alike of the giddiness and of the power of hearing. It is still more grave in the case of extreme severity from acute lesions, but these are so rare that they scarcely influence the general prognosis. In most other cases, the prognosis, although uncertain, is not definitely bad; improvement is exceedingly common, and in many cases goes on to recovery, if careful and persistent treatment can be secured.

TREATMENT.—In the treatment of aural vertigo we must recognise the double element in the pathology of the disease, the labyrinthine irritation, and the central instability induced by the irritation; the latter may be to some extent in excess of its cause. The central disturbance is lessened by bromide, which almost always reduces the tendency to vertigo, and, when this is slight, may remove it altogether. Twenty grains should be given two or three times a day, and the addition to each dose of a few minims of tincture of belladonna seems to increase its effect. Hydrobromic acid has been recommended, but must be converted into bromide in the blood, and only a quite inadequate dose of bromide can be given in the acid form. But it is also important to strengthen any weakness of the nervous system that can be traced.

The local irritability is commonly lessened by counter-irritation, whether the lesion is inflammatory or degenerative. The most effective counter-irritant is a small blister over the mastoid process. It is remarkable how rapid and marked may be the effect of a blister on all the symptoms, especially in recent cases. Drugs, unfortunately, have very little influence over morbid processes in the labyrinth, unless these are of a specific nature. Syphilitic inflammation can be readily removed, and the more common gouty changes can be lessened in a very marked degree by appropriate treatment. In the latter class I have several times known treatment remove not only the vertigo, but the tinnitus and the deafness, so that even the power of hearing through the bone, before completely lost, became normal. Purgatives, alkalies, and colchicum were the effective agents, but it is desirable to employ also counter-irritation, and at first to give bromide also, in order to lessen the morbid irritability of the centre. The bromide of lithium is a convenient salt to use in these cases, and some citrate of lithia may be added. It must be remembered, however, that degenerative changes occur earlier and progress more speedily in gouty subjects than in others, and hence, in such patients, the change in the labyrinth does not always prove amenable to the special treatment.

We can get little help from drugs in dealing with other chronic changes in the labyrinth. Charcot first suggested that agents which have a special action on this structure may possibly exert an influence antagonistic to the morbid process. With this object he gave large doses of quinine, so as to produce cinchonism. The patients were worse for the time being, but when the influence of the quinine had passed away, some of them were distinctly better. I have not found the effect of quinine so satisfactory as that of salicylate of soda, and I think that more good is done by giving it in moderate doses, five grains three times a day, than by administering it in such quantities as to produce toxic ear-symptoms. In several cases in which other treatment had failed, the moderate doses of salicylate rendered both the vertigo and tinnitus less troublesome. In all cases the conditions that lessen the tinnitus should be ascertained, and as far as possible their co-operation should be secured, since they may also exert a similar effect on the nerves concerned in causing vertigo.

It is important to treat any morbid influence that may co-operate in exciting the vertigo. The general health must be improved by tonics; exposure of the head to cold should be avoided, and the bowels should be kept open. Of special importance is the treatment of the dyspepsia which often co-exists, and has a very powerful influence in exciting the giddiness. As far as practicable, patients who are liable to vertigo should avoid stooping, and sudden movements of the head.

Nocturnal Vertigo.—Most persons, perhaps all, have been occasionally disturbed when falling asleep, or just after going to sleep, by a sudden sensation of falling from a height. Sometimes it is accompanied by a dream. I believe that this is really slight labyrinthine vertigo, due to spasmodic contraction of a tympanic muscle, which suddenly changes the pressure within the labyrinth. Those who wake up quickly during this sensation may distinctly hear the peculiar vibratory sound characteristic of intra-aural muscular contraction. It is identical in character with that which many persons may produce at will by contracting the orbiculares palbebrarum, and at the same time turning the eyeballs upwards. What muscle contracts is uncertain; it is perhaps the stapedius, which would suddenly lessen the pressure in the labyrinth. As soon as the sound of the contraction ceases, the sense of falling ceases also. These attacks may be prevented by a dose of bromide.

OTHER FORMS OF VERTIGO.

We have seen that it is exceedingly rare for definite vertigo to occur apart from aural symptoms, and it is certain that, in the majority of cases in which such vertigo has been ascribed to other causes, these have only had an exciting influence, and the symptom has been essentially due to the effect of unobtrusive labyrinthine disease, bringing the centre into an unstable condition. This is certainly true of the majority of cases of *gastric vertigo*, which was formerly thought to be so common; the mere presence of dyspepsia was regarded as a sufficient explanation of the giddiness. Certainly vertigo of purely gastric origin does not constitute more than 5 per cent. of the cases in which definite giddiness is the prominent symptom.* The giddiness met with in such cases is similar to that above described as met with in the aural form. The diagnosis rests on the fact that it distinctly follows stomach disturbance, and no other cause can be discovered. The treatment needed is, first, that for the gastric disorder, and secondly, the reduction of the central irritability by bromide.

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* I do not think it is quite certain that there is such a thing as definite vertigo of purely gastric origin. Thirty years ago 80 per cent. of cases of giddiness were supposed to be due solely to the stomach. But we now know that in 90 per cent. of the cases of definite giddiness a morbid state of the labyrinth is the real cause of the vertigo. It is possible that in the small remainder, of apparently gastric giddiness, there is some other influence that is the real cause, *e.g.* a morbid state of the semicircular canals causing no auditory symptoms, and so not to be detected save by its effects.

Vertigo occurs, in slight and vague form—a mere sense of unsteadiness—as a symptom of many morbid conditions, in which, however, it is subordinate to other more characteristic symptoms. Thus it is met with in anæmia, in hysteria, and in various conditions of nervous weakness. It is met with, although rarely, as part of an attack of migraine. It occurs also in the old whose brains are ill-nourished, in consequence especially of arterial degeneration, and also at the onset of various vascular lesions of the brain, of which it may be a premonitory symptom. The subjects of senile atheroma may suffer from occasional severe or constant slight vertigo, usually vague in character. The symptom, occurring in the old apart from distinct cause, is thus sometimes of serious significance, especially when it is paroxysmal and when headache is associated with it. It seems to be the result of the local interference with the blood supply, and suggests that an attack of hemiplegia is not unlikely. But care must be taken not to mistake for this the vertigo that is due to labyrinthine degeneration, senile or gouty. Many of those who suffer from this comparatively unimportant form are alarmed, and indeed injured, by groundless apprehensions of apoplexy. Vertigo is a prominent symptom in some cases of intracranial tumour, especially in tumours of the cerebellum or of the pons Varolii (see pp. 98 and 322), and it is very intense in lesions of the middle peduncle of the cerebellum.

Epileptic vertigo is a common form; the sensation, in all its varieties, occurs as the first symptom of the epileptic attack, and may be due to the inequality in the motor discharge in the two hemispheres that causes the initial deviation of the head and eyes, and sometimes a definite rotation of the body, but pronounced examples are occasionally met with.

Definite vertigo is occasionally met with apart from any recognisable morbid state, aural, gastric, or other, to which it can be ascribed. This form, in ignorance of its nature, has been termed *essential vertigo*. Most cases so described are labyrinthine, and those that can be regarded as independent of the common causes are so rare that we have little definite knowledge of their characters or nature.

One peculiar variety is, however, unconnected with the common causes of giddiness, and from its features may be termed *epileptoid vertigo*, if it is recognised that the name does not involve any actual connection with epilepsy. In this form sudden attacks of severe vertigo recur for months or years, with or without vomiting or vasomotor disturbance, but with perfect freedom in the intervals. In the suddenness of the attacks, their independence and recurrence, they resemble epilepsy, but they exhibit no tendency to pass into this disease. They differ, moreover, in their frequent excitation by fatigue, and in the long duration of the symptom, often for half an hour or even one or two hours. The sensation may be objective or subjective, or both; objects sometimes seem to rotate rapidly round a central point. The vertigo may or may not induce vomiting. Young adults have been the subjects in the few cases I have seen. The occurrence of the attacks is facilitated by fatigue and whatever depresses the nervous system. Mental tranquillity and physical rest are important elements in the treatment, and I have found the administration of nitro-glycerine in alcoholic solution, with a little hydrobromic acid, and sometimes also some bromide, particularly effective. It should be given regularly three times a day after food.

ASTASIA—ABASIA.

Not far removed from vertigo, at least in some of its features, is a peculiar symptom which has been recently described under this name, but is certainly much less rare than the unfamiliar character of the term might suggest. The sufferer from it, when walking, will suddenly fall to the ground (astasia), often forwards; it seems to him his legs give way. When sitting, he will suddenly bend forwards, his head dropping on his chest, and his body seeming to lose its power of support (abasia). In either case, in a minute or two, normal strength returns, and he is able to proceed as before the curious attack. There is never loss of consciousness. Rarely there has been a sensation of slight vague "giddiness" at the onset, but it is a symptom quite distinct from vertigo. Occasionally the fall has been preceded by a sensation as if struck by a hammer on the back of the neck. It occurs chiefly in persons past middle life, especially in the gouty and in those who have degenerated vessels. It has been thought to be of grave significance, but is certainly far less serious than the somewhat analogous paroxysmal dyslexia (see p. 116). In the cases I have seen the symptom has passed away under the influence of nervine and cardiac tonics, and the treatment and regimen suited for the constitutional condition. The symptoms must not be confounded with the "giving way of the legs" met with in hysteria, nor with the similar attack, accompanied by transient loss or obscuration of consciousness, of minor epilepsy.

NEURALGIA.

The word "neuralgia" means simply "nerve pain;" such pain may be due to actual disease of a nerve-trunk, by which its fibres are irritated, or it may occur without any organic lesion. It is true that the absence of such morbid change cannot often be proved by actual microscopical examination, but the transient character of the pain, and its migration from one part to another, frequently afford strong confirmation of the opinion that, in a large number of the cases of local nerve pain, the symptoms are not due to actual organic change. Two classes of neuralgias have been distinguished,—"symptomatic," in which the pain is a symptom of organic disease of the nerves; and "idiopathic," in which the malady, in the first instance at least, consists only in functional disturbance. Both of these forms have been called "neuralgia" by some writers. By others, however, the term is restricted to the idiopathic class, and this seems to be the only logical course. It is manifestly unreasonable to describe inflammation of nerves as "neuralgia" when it causes much pain and few other symptoms, and as "neuritis" when other symptoms predominate over pain. But the distinction of the two forms of nerve pain is often very difficult in practice. In some forms described as neuralgia the pain has certainly been generally the result of neuritis. This is the case, for instance, with sciatica, which, as stated in the account of the disease in the first volume, is generally an inflammation of the nerve. Yet not only has the common form of sciatica been described as a neuralgia, but its symptoms have been allowed to influence the description of neuralgia in general. In ascertaining the clinical history of neuralgia it is, therefore, of great importance that only cases should be used in which a primary organic lesion of the nerve trunk or centres can be excluded with reasonable confidence. But we have practically to include under the term those cases in which an organic process causes pains that persist longer than their cause, or extend far beyond its range of direct influence.

The subject of neuralgia is a very large one, so numerous are the forms of the disease, and so varied its characters. It will be most instructive to consider the general causes and symptoms of the affection, and also its general pathology, before describing its special varieties.

ETIOLOGY.—Neuralgia is essentially a disease of adult life. It is rare before puberty, and is not common in extreme old age, although, when the disease does commence late in life, it is often very severe and intractable. Most cases commence between twenty and sixty years. Children enjoy an almost complete immunity from true neuralgia, although very liable to certain headaches which do not come into this category. Women are more prone to neuralgia than men, but the degree of their liability has often been over-estimated, and the excess of females among the sufferers disappears in the second half of life. Moreover, the relative liability of the sexes is not the same in the several varieties. The tendency to neuralgia is often hereditary, although not so frequently as in the case of migraine. Anstie found evidence of heredity in only one quarter of his cases. Sometimes the inherited tendency is not special but general, indicated by the occurrence in ancestors or collaterals of epilepsy, insanity, and other neuroses. This is not surprising when we remember that the chief element in the affection seems often to be an excessive action of the central sensory cells, arising in them or induced from the periphery, but analogous to that which, in the motor and other cells of the brain, gives rise to the various forms of epilepsy.

The subjects of neuralgia often present a peculiar temperament. They are what is popularly called "nervous,"—excitable, often irritable, anxious, worrying over the trifling ills of life, sleeping badly, and are often extremely liable to headaches not distinctly neuralgic in character. In many patients the neuralgic tendency is deeply rooted; they suffer from neuralgia first in one situation and then in another during the course of years. Some forms are especially common in those who present the symptom of "hysteria," but whose sufferings from the pain are not the less genuine. Other varieties of neuralgia may also occur in such patients, by a coincidence which has only the connection of a common cause. The disease is more frequent in those of weakly constitution than in the robust, but the latter do not enjoy complete immunity. Among the constitutional relations of neuralgia, those to rheumatism and gout are especially important. The connection with rheumatism is often conspicuous, and is seen in several aspects. Persons who are liable to rheumatism of the fibrous tissues sometimes suffer from pains which have both rheumatic and neuralgic characters,—not specially related to the nerves in situation, and yet paroxysmal and unconnected with movement. Such pains are especially frequent in the limbs and back. Women who suffer from rheumatoid arthritis are often also liable to true neuralgias of great severity. A young lady, for instance, suffered for several months from severe paroxysms of pain in one shoulder and the dorsal spine, apparently neuralgic; these ceased, and she was immediately attacked by subacute rheumatoid arthritis. Lastly, both rheumatic affections and neuralgia are certainly sometimes due to gout, and probably not unfrequently to inherited gout.

Among the exciting causes of neuralgia, as among those that are remote, impairment of general health takes the first place. The affection may be excited by any kind of debilitating influence: overwork of mind or body, over-lactation, prolonged fatigue, and anæmia of every degree and causation, are frequently met with as its immediate antecedents. Certain forms of sensory fatigue, as over-stimulation of the eyes, sometimes seem to have a special influence. Severe emotion, or its physical analogue, mechanical concussion, are also occasional exciting causes; the former is often combined with the latter. As examples of the influence of emotion two cases may be mentioned, one of slight, the other of severe neuralgia, thus induced. A lady was intensely distressed after parting with her husband, who was going to America. She felt on the point of bursting into tears, and as if the tears would give her relief. Her sister said, "Do not cry; you *shall not* cry." By an effort she succeeded in restraining her tears, but was immediately conscious of a sense of intense pressure above the eyebrows, and a few days later severe supra-orbital neuralgia came on upon the left side, and lasted for several weeks. A girl of eighteen was much startled and alarmed by the unexpected discharge of a gun close beside her. The same evening facial neuralgic pain came on, and continued for five years in most violent paroxysms, sometimes on one side, sometimes on the other. In this connection it may be noted that the fifth nerve is especially related to emotion, both by influencing the secretion of tears and also as the sensory nerve of the chief region of emotional display—the face.

No single actual excitant of neuralgia is so frequent as exposure to cold, sometimes general, sometimes local and affecting the part in which the neuralgia is felt. Valleix found a history of exposure to cold in one third of his cases. Cold may not only produce neuralgia, but may also excite paroxysms of pain when the neuralgia is due to some other cause. Another frequent cause is the irritation of nerves, especially near their peripheral distribution, which induces pain extending far beyond the area supplied by the irritated nerve. A common example is the wide-spread pain that may result from the irritation of a carious tooth; the pain may extend into other divisions of the fifth nerve, and even into the region of the cervical plexus. Moreover, the pain may be felt only or chiefly in some other region than that in which it is produced. Thus I have known severe neuralgia confined to the second division of the fifth to be due to a carious tooth in the lower jaw, and cease entirely when this was extracted. Every form of neuritis, except that of purely motor nerves or branches, may have, as its sequel, pain of long duration, which may be regarded as a secondary neuralgia. Brachial and sciatic neuritis, either primary or traumatic, local or migratory, are common causes of this form. Another is the peculiar neuritis that is manifested by herpes zoster. The severity and duration of the subsequent pain is chiefly serious in late life, when it may continue even for one or two years, and be most distressing in severity. Traumatic lesions of nerves without any preponderating neuritis, constitute another cause, and often give rise to pain that is extremely obstinate.

Toxic influences often induce neuralgia. The most frequent are alcoholism, lead-poisoning, and the presence of an excess of sugar in the blood. But these causes also produce neuritis, of which pain and tenderness may be the only evidence, and it is often difficult to exclude actual inflammation of the nerve. At the same time, an agent which causes neuritis may sometimes merely irritate the sensory structures and produce neuralgia. This statement is also applicable to gout, the influence of which has been already mentioned. Both neuritis and neuralgia may unquestionably result from acquired and inherited gout. Malaria is another powerful cause, but its influence is seldom seen in this country, except in those who have been abroad. Neuralgia is an occasional sequel of various acute diseases which depress the nervous system, but it follows none so often as influenza. It may then occur in any locality, sometimes after a trifling exciting influence, and may last for weeks or months; in some cases it probably succeeds an initial neuritis. The syphilitic poison has been thought, by some, to cause true neuralgia during its active stage, but the evidence is not strong. Occasionally, those who have had syphilis long before are the subjects of paroxysmal pains, in character and course identical with those of tabes, but yet present no other indication of this disorder. It is possible that this is a "tabetic neuralgia," due to degeneration in the sensory nerves, such as occurs in tabes, but sparing the muscle nerves, in which it causes ataxy.

SYMPTOMS GENERALLY.—The great symptom of neuralgia is pain, spontaneous, paroxysmal, and felt in certain regions of nerve distribution. It is usually unilateral; when bilateral it is almost always symmetrical in distribution. Usually the pain is constant in seat for a time, it may be for many years. In other cases it changes, now in one part, now in another. Thus a girl aged fifteen had suffered for two years from paroxysms of intense pain in various parts, arms, legs, back, different parts of the head, and occasionally universal.

The pain is never constant in degree; there are paroxysms with intervals of complete freedom, or there is a slight continuous pain with intense exacerbations. Continuous pain may be merely a dull ache, but it is generally acute and sharp during the paroxysms, and is described as "darting," "stabbing," "boring," "burning," &c.; often the sufferer can find no words to express its exact character. The sharp pain generally has a darting character. A series of sudden sharp pains occurs every few minutes; the series of successive pains constitutes a paroxysm, and a series of paroxysms an attack. The intervals between the attacks present extreme variation, and are sometimes remarkably long in proportion to the severity of the pain. Thus one patient will have attacks daily during several years, while in another (as in an actual instance) intervals of many months separate groups of attacks of most intense pain, each group lasting only a few days. In such paroxysms it is usual for the pains to commence suddenly, but they are rarely as severe at first as they subsequently become. Sometimes a peculiar sensation, such as throbbing, heralds each attack of pain. The attacks gradually increase in intensity, and in each attack the separate paroxysms may present a characteristic augmentation and decrease.

The pain is seldom referred to the skin; usually it is more deeply seated, and often corresponds to the position of a nerve-trunk and its branches—a fact of some diagnostic importance. The throbs of pain sometimes, but seldom, coincide with the arterial pulsations. There is often a darting movement of the pain, usually towards the periphery—centrifugal; less commonly from the periphery—centripetal; still less commonly the pain darts alternately in both directions. Sometimes it seems to be localised in a single point, and then has usually a boring character. When most intense the darting pain radiates to other nerve-regions adjacent to that in which it is chiefly felt.

The duration of each attack varies according to the number of paroxysms and their length. Rarely there is a single brief, intense pain; more often an attack lasts several hours. In the intervals there may be slighter pain, or a peculiar sensation in the part, or freedom. The intervals vary from a few hours to several months. Often periodicity exists; it may be exact in malarial cases, in which the pain may commence at the same hour each day, or at intervals of two or three days. Now and then the periodicity is exact in cases that are not malarial. The pain may be worse at the catamenial periods, and may even occur only at those times. When there is continuous pain in the intervals between the paroxysms, it is moderate in degree, but often troublesome by disturbing rest.

The paroxysms and attacks are often induced by certain influences, external or internal, such as by exposure to cold, sometimes by warmth, by movement, posture, or emotion. In some cases there is remarkable sensitiveness to atmospheric influences. When the paroxysms occur at regular intervals, an influence that will induce the pain when it is "due," may be powerless immediately after an attack. During a paroxysm, the influences that will bring it on usually intensify the pain. Movement is especially influential in the neuralgias of the fifth nerve; the slightest motion of the jaws may induce the pain. A touch on the skin may have the same effect; nevertheless in many cases, although slight pressure increases the pain, firm pressure gives distinct relief; even when mere contact with the skin causes an exacerbation, rough rubbing may distinctly relieve the suffering. This difference, however, is not always to be observed. In some cases alcohol, even in small quantities, invariably intensifies or induces the pain; in other cases it gives relief.*

This increased sensitiveness of the skin is a very common accompaniment of the pain. It may involve all forms of sensation, although thermic impressions much less commonly occasion pain than does a touch. Sometimes the tactile impression seems to be felt as pain; more often it excites an increase in the true neuralgic pain. The hyperæsthesia, or hyperalgesia, is usually limited to the region in which the spontaneous pain is felt. When this pain follows the course of a nerve it is commonly most intense at certain spots, and at these places pressure may cause a special increase in the suffering. In the intervals between the paroxysms these spots may remain tender, and pressure upon them may induce a paroxysm. They are not usually present until the disease has lasted for some time. In recent cases, and when the attacks occur only at long intervals, although there is no persistent tenderness, the pain may be increased during the paroxysm by pressure on certain places. The tender points were first studied by Valleix, and hence are often called after him. They are tolerably uniform in their position, and for the most part correspond either to the place at which a nerve-trunk emerges from a bony canal, passes over a hard structure, or passes through a fascia to become superficial, or to the point of division of a nerve-trunk, or to an anastomosis of two nerve-trunks. They are present in about half the cases; when absent there is sometimes ill-defined tenderness in certain areas.

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* Very curious facts are sometimes met with in regard to the induction of the pain. Thus in one patient intense fronto-occipital neuralgic pain was excited by every act of defæcation. This action, in some cases, has a very peculiar influence on the nervous system, and so has micturition, as the familiar shiver shows. I have known micturition to be attended with a moment's loss of consciousness.

There is occasionally tenderness of the vertebral spine corresponding to the origin of the painful nerve, the *point apophysaire* of Trousseau. It is probable, as Anstie pointed out, that the relation of this to neuralgia has been exaggerated. The tenderness of certain vertebral spines is common apart from neuralgia, and there is not always a close correspondence between the position of the spinal tenderness and the seat of the neuralgia. In trigeminal neuralgia, for instance, there may be tenderness of the cervical spines.

Other sensory disturbances are occasionally observed in neuralgia. The onset of the pain is sometimes preceded by numbness, tingling, &c., in the affected area, but less frequently than in neuritis. Occasionally the attack of pain is followed by transient anæsthesia. Persistent diminution of sensibility is only met with in cases of "symptomatic neuralgia," in which there are structural changes in the nerves. Increased sensitiveness to pain (hyperalgesia), in the whole area of the neuralgia, is not uncommon. Vomiting is rarely associated with simple neuralgia, although so common in migraine, in which the pain may bear a superficial resemblance to that of neuralgia. But I have met with vomiting in two cases of neuralgia; one, bilateral, in the anterior branches of the cervical plexus, and the other in the two upper divisions of one fifth nerve. Severe attacks in women often cause hysterical symptoms as the pain is subsiding.

Muscular spasm may be excited by the acute paroxysms of pain, evidently in a reflex manner. It is usually confined to the motor nerve related to that which is the seat of the pain, but sometimes spreads to adjacent areas, very rarely passing into a general convulsion. In a case of cranio-spinal neuralgia each paroxysm was attended by opisthotonos so severe that the patient rested on the head and the heels. The exacerbation of the pain by movement may lead to temporary diminution of mobility, partly voluntary, partly of inhibitory origin.

Trophic and vaso-motor disturbance sometimes results from the deranged nerve-influence. Skin eruptions are rare; herpes is seldom, if ever, a consequence of true neuralgia, although neuralgic pain so often accompanies and follows herpes. The hair of the part may undergo changes: it may lose its pigment, fall off, or very rarely overgrow. Anstie observed temporary greyness of a lock of hair after each attack, followed at last by permanent change.* I have seen, in a girl of eighteen, loss of pigment in the hair above and behind each ear, following neuralgic pain there of some months' duration. Vaso-motor disturbance often accompanies a paroxysm. The first effect of the pain is usually to cause a constriction of the vessels of the part, but this is often followed by their relaxation; flushing of the skin results, and the throbbing of the arteries may considerably intensify the pain. The arterial dilatation may be general, and be demonstrable by sphygmographic tracings (Anstie). In one case of trigeminal neuralgia all the veins of that side of the face became distended during the paroxysm, and as the pain subsided pallor replaced the flushing. The local vascular disturbance may cause local sweating or local œdema, or even erythema, sometimes mistaken for erysipelas. The œdema thus produced is occasionally considerable; I have known each attack of cranial neuralgia to be accompanied by great swelling of the whole scalp, due to this condition, which slowly disappeared some hours after the cessation of the pain. In another curious case, attacks of pain in the tongue and face were attended by swelling of each part, which usually came on during the night, and sometimes occurred with very little pain. Repeated attacks of such vaso-motor disturbance may lead to permanent dilatation of the vessels of the surface, and, after a time, to thickening of the cellular tissue, periosteum, and other structures.

PATHOLOGY.—Few questions have been the subject of more controversy than the pathology of neuralgia. The difference of opinion is largely due to the different senses in which, as we have seen, the word has been used. The problem of pathology is, What is the nature of nerve-pain that has no known organic cause?

In neuralgia we have two symptoms; first, spontaneous pain, and secondly, "hyperæsthesia" (more properly hyperalgesia), *i. e.* the production of pain by sensory impressions that are not usually painful: the former includes the latter, and therefore must be first considered. Spontaneous pain means the action of sensory nerve-elements apart from local external stimulation. The pain corresponds to certain peripheral nerve-areas, and we must therefore look for its cause to the elements constituting a peripheral nerve-structure. These are the nerve-fibres, their peripheral end-organs, and the central cells with which the fibres are connected. To which of these can we ascribe a functional activity independent of external stimulation? We know nothing of a capacity for such action on the part of nerve-fibres. They possess a limited power of transforming external energy into nerve-force, which constitutes their "excitability," and they "conduct" nerve-force; but there is no evidence to show that, apart from external influences, they are capable of producing nerve-force. Nor are any facts known which would suggest that the peripheral end-organs of the sensory nerves are the seat of the primary disturbance in simple neuralgia. It is true that, in tabes and degenerative peripheral neuritis, changes in the nutrition, and sometimes in the structure of the nerve-endings seem, in many cases, to cause the acute pains of these disorders. The function of these structures, transforming mechanical into nerve energy, must involve a delicate organisation, susceptible of almost spontaneous over-action; but the features of true neuralgia differ from those produced by disease of the nerve-endings. We are thus led to look to the central terminations of the nerve-fibres as the source of the pain in idiopathic neuralgia.* The fibres end in nerve-cells, and nerve-cells are the elements chiefly capable of the production of nerve energy, in apparent independence, and certainly in excess, of the stimuli applied to them. When an external cause (*e.g.* an injury of the nerve) gives rise to pain, the sensation is due to the stimulation of these cells, and the pain which results from their spontaneous "discharge" is necessarily referred to the region from which their fibres convey impressions.

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* A very remarkable case has been recorded by Raymond, in which, at the time neuralgic pain in the head was most intense, all the hair of the patient (a woman aged thirty-eight) changed colour from black to red, and in a few days to white, and then, in the course of fourteen days, fell off ('Revue de Méd.,' Sept., 1882).

The sensory fibres end in two sets of cells, those of the ganglia on the posterior roots, and those within the spinal cord, chiefly in the posterior cornua. There is no direct evidence to show which of these series of cells is concerned in neuralgia. But we know nothing of any sensory function of the ganglia, and we are therefore justified in looking to the nerve-cells within the cerebro-spinal axis as the seat of the morbid process.

This conclusion is supported by the symptoms of many cases of neuralgia. Especially significant are the following—(1) The fact that the pain may occupy adjacent parts of several nerve-regions. For instance, in one patient the pain extended on both sides from the seventh cervical vertebra, over the whole occiput and vertex to the coronal suture. (2) The phenomenon of radiation of slighter pain into adjacent nerve-regions during severe paroxysms. (3) The phenomena of reflex neuralgia, in which the pain is felt in another region than that of the nerve irritated. All these are explicable only on the theory that the morbid action, felt as pain, is in the central cells, which are no doubt connected according to the relations of the surface regions from which they receive impressions, and to which their disturbance is subjectively referred.

The same conclusion, the central nature of neuralgia, is also indirectly confirmed by facts of pathology of another kind which prove that pain of neuralgic character may be produced by an organic lesion in the grey matter which is here supposed to be deranged in idiopathic neuralgia. The lesion, for instance, involving part of the sensory nucleus of the fifth nerve, shown in Fig. 53 (B x), p. 69, caused severe neuralgic pain in the face.

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* The central theory has been adopted, amongst others, by Vulpian, Anstie, Clifford Allbutt, and Vanlair, although the theories of these authors differ somewhat in their details.

If, therefore, we regard idiopathic neuralgia as the result of the over-action, the "discharge," of the nerve-cells constituting the proximate centre of the nerve, the question still remains—to what is this discharge due? It is often ascribed to hyperæmia of the centre, due to dilatation of its vessels. The possibility of this cause cannot be denied, but neither can its efficiency be proved. It is an hypothesis, moreover, which only solves one problem by the introduction of another. Vaso-motor disturbance means the deranged action of the vaso-motor centre for that territory, and it is as difficult to explain the disturbed action of the vaso-motor cells as of the sensory cells. All cells possess the power of evolving force; the discharge of the sensory cells is that alone of which we have evidence, and it seems unjustifiable to assume the intervention of other cells for its production. But it is highly probable that secondary vaso-motor disturbance results. We know that in all organs vascular dilatation attends functional activity. If the cerebral cortex is stimulated by electricity, local dilatation of vessels quickly follows. It is probable that such secondary central hyperæmia may result from the discharge of sensory centres, and when established, may increase the disturbance.

The over-action of the cells in idiopathic neuralgia has been spoken of as "independent." It is independent so far as our means of observation go. But we cannot tell to what extent the unstable cells may be excited to discharge by stimulation of which we are unconscious. We know that some influences of this kind (cold or pressure) are effective in exciting attacks. It is highly probable that the afferent impressions which we perceive bear but a small proportion to those of which we are unconscious; these, continuous or intermittent, are due to slight cutaneous impressions, to the movement of the blood, and to the nutritional processes in the tissues. It is quite possible that such afferent impulses, too trifling to affect our consciousness even when aided by attention, may excite the discharge of the unstable cells. That a strong sensory impression (*e.g.* painful pressure on the nerve) may sometimes relieve the pain is quite in harmony with familiar facts as to the effect of sensory stimulation; a strong stimulus may inhibit the reflex action that may be excited by a slighter stimulus in the same nerve-region; sometimes pressure may arrest exciting impulses. These two facts deprive of much of its force an objection which has been often urged against the central theory of neuralgia, that the disease may sometimes be permanently cured by division of the nerve; the significance of this will be considered in connection with treatment.

We are accustomed to think of the sensory nerves as distributed chiefly to the skin, but the pain in neuralgia is rarely confined to the skin. It appears to the sufferer to be more deeply seated, and often corresponds in position to the nerve-trunk and branches. The fibrous sheaths of both are abundantly supplied with nerves,—the *nervi nervorum*,[*] which ramify and end in the sheath, without penetrating the interfascicular septa. If a nerve is compressed (as the ulnar at the elbow) the first sensation experienced is a pain at the spot, from the stimulation of the sheath-nerves; if the pressure is continued, there is also a sensation (tingling) referred to the peripheral distribution of the nerve in the hand, due to the stimulation of the fibres of the nerve itself. In neuralgia, the central cells of the sheath-fibres seem often to be disturbed in function more than those of the fibres of the nerve itself; the pain is referred to the nerve-trunk rather than to the skin.

The fact that the pain seems to dart along the nerve must be due to the spread of the discharge in the centre in a certain order from cell to cell. We cannot at present say precisely on what this depends, or why the pain seems sometimes to dart towards and sometimes from the periphery.[†]

The irradiation of the pain, in severe attacks, to neighbouring nerve-areas is clearly, as already mentioned, a central phenomenon. An intense discharge always tends to spread to other connected cells, in proportion to its intensity, as the phenomena of epilepsy abundantly illustrate.

The origin of the tender points is obscure, and has been the subject of much speculation.[‡] Their localisation to the places at which nerves emerge from deeper structures, or divide, suggests their dependence on mechanical causes. Accidental pressure, and traction in movement, will have most influence on the nerves at such places, and cause there a greater degree of stimulation of the nervi nervorum (Vanlair). Many phenomena of neuralgia suggest, moreover, that a neuralgia which is at first purely central may not remain so. We have seen that the pain often causes secondary vascular disturbance in the territory of the nerve. It is most unlikely that such disturbance, conspicuous in the skin, is confined to the surface. It probably involves also the deeper structures, and especially the nerve-sheaths, in which the pain is especially localised. Such secondary vascular disturbance, and the tissue changes to which it ultimately leads, must constitute a source of irritation of the nervi nervorum, and, in a truly "vicious circle," must intensify the malady, which, at first central, may be thus, at last, peripheral also. It is probable that this mechanism takes an important share in the production of the tender points, and is also the cause of the intractability of some neuralgias.

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[*] Sappey ('Journal de l'Anat. et de la Physiologie,' vol. i, 1868, p. 47) has demonstrated their existence in the sheath of the optic nerve, and the fact mentioned in the text admits of no other explanation than the existence of similar nerves in all nerve-sheaths, which has, indeed, been demonstrated by Horsley ('Proc. Roy. Med. and Chir. Soc.,' 1884, and Appendix to Marshall's 'Bradshaw Lecture,' 1887).

[†] An ingenious hypothesis has been formulated by Vanlair, which may be mentioned. According to Pierret there is a relation between the length of a nerve-fibre and the size of its cell. The sheath-nerves will vary in length according to the distance from the centre at which they terminate. If the cells discharge in the order of their size, beginning with the smallest, the sensation will seem to dart centrifugally. For the apparently centripetal direction a still more complex hypothesis has been suggested. Most nerves contain recurrent fibres, which proceed from adjacent nerves (Magendie, Arloing, and Tripier). These recurrent fibres ascend the branches and trunk, but all cease before the foramina of exit are reached. If these end in the nerve-sheath, the same hypothesis applied to the recurrent fibres will serve to explain the centripetal darting; the longest fibres, which end nearest the centre, will have the largest cells, and discharge last. Unfortunately we do not know that any of the recurrent fibres end in the nerve-sheath, and if they do, it is probable that their central connection is the same as that of the direct fibres. It seems more probable that the arrangement of the cells in the centre depends on the distribution of the fibres in the sheath, and that this arrangement, and not the size of the cells, determines the order of the discharge, which may traverse the centre in opposite directions in different cases, just as in one epileptic an aura may pass down, and in another up, the arm.

[‡] The hypothesis (of Cartaz and others) which connects these tender points with

Not only is it probable that peripheral disturbance takes part in the pathogenesis of central neuralgia, but it is certain that central disturbance is concerned in some neuralgias of peripheral origin. A traumatic cause, an injury to a nerve-branch, may induce pain far wider in area than the distribution of the injured branch, or even of the nerve from which it comes. In some cases, again, the pain is felt not in the area of the nerve injured, but in that of some other nerve (reflex neuralgia). An interval usually elapses after the injury before the pain is felt. Lastly, in some traumatic cases, division of the nerve does not cure the neuralgia. These facts can only be explained by assuming that the chief cause of the pain is a morbid state of the central cells, excited by, but to some extent independent of, the peripheral lesion. Doubtless in all neuralgias of "symptomatic" character the symptoms depend, in varying degree, on an induced central disturbance.

Various Forms.—Cases of neuralgia differ much according to their situation, character, and cause, and hence it is necessary to describe in some detail the varieties of the disease. According to situation, we have to consider separately those which occupy the head, neck, arm, trunk, and leg. According to character, we have "epileptiform neuralgia," and "reflex" or "sympathetic neuralgia;" while of those which depend on special causes the most important are the traumatic, herpetic, anæmic, malarial, syphilitic, and diabetic forms.

It is important to remember that neuralgia is often not confined to a single nerve. Those who are liable to the affection in a high degree

the distribution of the recurrent nerves, rests on too many unprovable assumptions to deserve detailed description.

sometimes suffer from neuralgia in many situations, simultaneously or in succession.

Varieties depending on Situation.—Neuralgia of the Fifth Nerve. Trifacial or Trigeminal Neuralgia; Tic Douloureux; Prosopalgia.—Under these various designations the most common form of neuralgia has been described. Neuralgia of the fifth is probably more frequent than all the other varieties together, and it presents, in most typical form, the characteristic symptoms of the disease. Nor is this surprising when we consider that the fifth is incomparably the most important nerve of common sensibility in the body.

The causes of this form are all those that have been described in the section on etiology; indeed, the general history of neuralgia is, to a large extent, based on the symptoms of this variety. It is equally common on the two sides. The seat of the pain may be in any of the three divisions of the nerve; and it more commonly occupies one or two of the divisions than all three. The tender points are often well marked, and in them the pain has its chief intensity.

Neuralgia of the *first division* is felt chiefly in the supra-orbital branch, and hence is often called *supra-orbital neuralgia*. The frequency with which it was formerly due to malaria has left for it the popular name of "brow-ague," although this cause is now rarely operative in this country. The pain radiates from the supra-orbital notch over the anterior half of the head, and is often felt in the eyelids and even in the eye, and in the side of the nose. The most important tender points are the *supra-orbital* just above the notch or foramen of that name, a *palpebral* in the outer part of the upper eyelid, a *nasal* at the emergence of the nasal branch at the lower edge of this bone, and sometimes an *ocular* within the eyeball. Pain felt just above the eyebrows is sometimes due to a morbid state of the frontal sinuses, but pain from this cause is generally double, and is often secondary to coryza. The lining membrane of the sinus is supplied by the fifth nerve, and it has been conjectured that the pain occurs when the small opening of the sinus into the nasal cavity becomes closed. Seeligmüller thinks that this is the cause of the pain even in malarial cases. But the nerves of the sinus (or their centres) seem to be particularly obnoxious to certain influences, as is shown by the peculiar pain, evidently referred to these sinuses, which many persons experience after eating ices. We cannot, therefore, conclude that because the pain occupies this situation, it is necessarily due to a local cause. The supra-orbital region is a not uncommon seat of localised pain that is apparently neuralgic. This does not follow the course of the nerves, but it may be felt sometimes over one eye, sometimes over both.

Ocular Neuralgia.—The eyeball is an occasional seat of neuralgic pain, often of considerable severity. This is sometimes associated with some error of refraction, especially hypermetropia, but occurs also independently of any abnormality of the eye itself. Either one or both eyes may be affected. The pain may occur spontaneously, or may be brought on by use of the eyes; it is not often accompanied by photophobia. When severe, there is occasionally dimness of sight, apparently of inhibitory origin, and the amblyopia may be accompanied by a peripheral restriction of the field of vision. Ocular neuralgia may exist alone, or be associated with pain in adjacent parts, and sometimes with pain that extends far beyond the limits of the fifth nerve. Bilateral pain sometimes passes from the eyes over or through the head to the occipital region, and even down the neck. Anæmic girls often complain of a peculiar dragging pain at the back of the eyes, increased by their use. Ocular pain is often associated with rheumatism; the subjects of rheumatic iritis may be liable to pain in the eyeballs, which seems to be neuralgic in character.

In neuralgia of the *second division*, *infra-orbital neuralgia*, the pain occupies the area between the orbit and the mouth and extends over a great part of the cheek, and to the ala nasi. The chief foci of pain and tender points are an *infra-orbital*, at the emergence of the nerve beneath the orbit; a *nasal*, at the side of the nose; a *malar* over the most prominent part of that bone, and a *gingival line* below that bone, along the line of the gums of the upper jaw: very rarely there is a point in the palate or in the upper lip. The most acute pain is often felt only in one portion of the nerve, as, for instance, the side of the nose; but it usually radiates, in a slighter degree, over a wider extent.

When the *third division* is affected, the pain often extends over a large area, occupying the parietal eminence and the temple, the ear, the lower jaw, and the tongue. The chief tender points are the *inferior dental*, at the foramen of that name; the *temporal*, in the posterior part of the temple on the auriculo-temporal branch; it may be a little lower down, just above the zygoma, in front of the ear, and is a very common focus. The *parietal*, over the parietal eminence, is common to this and to occipital neuralgias. Sometimes there is a focus of pain in the tongue. Separate portions of this branch are sometimes affected alone, especially the inferior dental and the auriculo-temporal. A boring pain limited to the temporal point is especially common. Most intense neuralgia is sometimes confined to the lingual branch. Occasionally a tender point exists in the cervical spines at the first two or at the fifth; its exact cause is obscure.

The pain in trigeminal neuralgia is often peculiarly intense, and presents every variety. It may radiate from one part of the fifth nerve to the next, and even to other nerve-regions. Thus, in one case of violent neuralgia of the second division of the fifth, the pain often radiated to the occipital region, and sometimes to the shoulder on that side. The effect of cold and contact in exciting the pain are well marked, and it is often increased by movement of the face or jaw, so that, in severe cases, mastication may be impossible, and it may be difficult to give sufficient food. If the ear is the seat of pain, either alone or with other parts, the attack may be accompanied or followed by auditory hyperæsthesia. When the pain is very acute and sudden, reflex muscular spasm may occur in the face (the "tic convulsif" of the French). Paralytic phenomena are rare, but transient paralysis of the third nerve has been observed to follow each paroxysm of pain in the supra-orbital branch. Sometimes paroxysms of severe pain in the fifth nerve are accompanied by subjective flashes of light, especially when the eyeballs are the seat of pain. The vaso-motor disturbance already described is frequently seen, flushing, sweating, permanent dilatation of the vessels (often conspicuous on the eye), salivation, increased secretion of mucus in the nose, lacrymation. Trophic disturbances occasionally occur,—acute, as erythema, or chronic, as thickening of the periosteum, loss of hair, or local greyness. Even unilateral atrophy of the face has been met with. It is probable that in many of these cases the pain has been the result of actual changes in the nerves. The course of the fifth in the base of the skull, through the membranes and bony foramina, exposes it to damage from many morbid processes, and causes its fibres to suffer when there is any inflammatory swelling of the sheath.

Occasionally, migratory pains are felt in various parts of the scalp, sometimes on one side, sometimes on the other, without any distinct relation to the nerve-trunks. There may be tenderness of the skin during and after the paroxysms of pain. This form is sometimes more closely allied to rheumatism than is ordinary neuralgia. Rarely there is neuralgic pain over the whole scalp at the same time, so that, as one patient expressed it, there is "a cap of pain on the head." Pain at the vertex is a common form of headache, sometimes closely allied to neuralgia, and it may alternate with characteristic neuralgic pains in other situations.

Cervico-occipital Neuralgia.—The pain is felt in the region of the neck supplied by the first four cervical nerves, and in the posterior portion of the head, chiefly along the course of the great occipital nerve. Thus the pain may extend over the greater part of the neck as well as over the head, as far forward as the parietal eminence and the ear. It is occasionally confined to the posterior branches, extending over the back of the neck and occiput. The most important tender points are (1) about midway between the mastoid process and the spine, at the point at which the great occipital nerve becomes superficial, (2) over the branches of the cervical plexus between the sterno-mastoid and trapezius, and (3) just above the parietal eminence, the focus common to occipital and trigeminal neuralgia. Fusion of these two forms of neuralgia occurs, not only above but also below, where the distribution of the cervical nerves blends with that of the third division of the fifth over the lower jaw. A primary cervical neuralgia may extend into this region of the fifth; doubtless the centres blend as does the distribution. It is probable that cervico-occipital neuralgia is more often bilateral than any other form, especially when confined to the occipital region. I have known most severe bilateral neuralgia to be limited to the anterior cervical region, from the jaw to the upper part of the thorax on each side. The pain in cervico-occipital neuralgia is rarely intermitting; there is more or less dull constant pain with occasional exacerbations, less violent than in the trigeminal form. The scalp may become extremely tender, so that, during the pain, the patient cannot bear the hairs to be touched. This form of neuralgia is not common, and Anstie believed that it occurs especially in those who have suffered from other forms.

Cervico-brachial and brachial neuralgia include those forms in which the pain is referred to the region supplied by the four lower cervical and the first dorsal nerves. The pain may be felt in the lower and posterior part of the neck, or any part of the arm and hand, but is commonly most intense in the axilla, at the brachial plexus, and along the course of the ulnar nerve. The region of the last is a very frequent seat, but sometimes the pain is referred to other nerves. It is commonly increased by movement, and may render the arm almost useless. It is often excited by writing, and this may give rise to an erroneous impression that it is connected with the act of writing. Some severe forms of brachial neuralgia, indeed, have their origin in a sensory occupation-neurosis, but this variety has been separately described (p. 710). The most common tender points in brachial neuralgia are the *axillary*; the *circumflex*, at the posterior border of the deltoid; a *superior ulnar* behind the elbow, and an *inferior ulnar* in front of the wrist. The latter is the most frequent of all the brachial foci. Others occasionally met with are the *vertebral*, by the side of the lower cervical spines; a *scapular*, at the inferior angle of that bone; an *external humeral*, on the outer side of the arm three inches above the condyle, over the musculo-spiral nerve; and a *radial*, in the lower and outer part of the forearm. There is usually some constant pain in addition to the acute paroxysms. Occasionally it may radiate to the side of the chest, and then, if on the left side, may simulate angina pectoris. The pain is almost always intensified by movement. Trophic disturbance in the arm is very rare in cases of true neuralgia, and, indeed, probably always indicates neuritis. When the pain starts from the fingers, it may begin with some sensation other than pain, such as tingling, which changes to pain as it passes up the arm. In some patients with brachial neuralgia the arm is peculiarly liable to be the seat of tingling at night. Brachial neuralgia is not often due to diathetic causes, with the exception of rheumatism, with which it is often associated even when there is no suspicion of neuritis. On the other hand, it is more frequently than any other the result of injury. Probably many cases of supposed neuralgia are really cases of neuritis of the brachial plexus, which may arise by migratory inflammation. But true neuralgia of the arm, widely spread, may be set up by a slight injury, as a blow, which does not apparently cause neuritis. Brachial neuralgia is occasionally associated with neuralgia of the fifth, and this when there is no connecting pain in the neck.

Trunk Neuralgia.—Of the neuralgias of the trunk, we have, first, the *dorso-intercostal* forms, which occupy the intercostal nerves, from the third to the ninth, characterised by pain coursing along the intercostal spaces or parts of them. It is sometimes bilateral and symmetrical. There is usually a constant dull pain, with acute stabbing exacerbations, but sometimes the continuous pain exists alone, or the sharper pains are excited only by movement, respiratory or other. There are foci of intensity and tender points at the emergence of the three branches of the intercostal nerve,—beside the vertebræ, near the middle line in front, and midway between these two points in the mid-axillary line. Intercostal neuralgia is most frequently due to cold, or to injury, such as a contusion. It is sometimes extremely obstinate and of long duration.

The thoracic wall is also the seat of more trifling neuralgic pains; one of these is pleurodynia, which differs from true intercostal neuralgia in being usually localised at some spot not corresponding to the course or exit of the intercostal nerves. It appears to be a true neuralgia, distinguished from myalgia by the fact that it is local, very acute in character, and is excited by expansion of the thorax rather than by lateral movements of the trunk. The theory that it is a neuralgia of the pleural nerves has much probability. Another common neuralgia is the infra-mammary pain of anæmic women. Very limited in position, it is more or less constant, and is rarely increased by respiration to such an extent as to interfere with the free expansion of the thorax. The relation between intercostal neuralgia and pulmonary trouble is a difficult subject, on which satisfactory facts are difficult to obtain, and few observers have ventured to corroborate the statement of Woillez that acute intercostal neuralgia is always accompanied by a secondary congestion of the lung. It is more probable that when this association exists, the true relation is the reverse. The intercostal nerves are frequently also the seat of herpetic neuralgia.

The neuralgias that occupy the lower half of the trunk have been grouped as *lumbo-abdominal.* The pain has a course similar to that of the intercostal form. Foci of pain and tender points are found at the back, beside the vertebræ, over the posterior branches; at the middle of the iliac crest (*iliac point*); at the lower part of the rectus (*hypogastric point*), while sometimes there is in the male a *scrotal*, and in the female, a *labial point.* These pains are often bilateral, and may change their position from time to time. They are generally acute pains, but now and then have a constricting character, like the girdle-pain of organic disease, but distinguished from them by their mutability. Lumbo-abdominal neuralgias seem to be sometimes secondary to disease of the pelvic organs, especially in the female. Neuralgia in the penis has been thought to result from masturbation. It may also be due to lithæmia, and from this cause I have known it to be most severe and long continued.

The *spinal column* is a very common seat of neuralgic pain, especially in weakly women and after concussion of the spine. It constitutes one of the most troublesome of the many pains of hysteria, and one of the most enduring consequences of railway accidents. The pain, in most cases, is felt through a considerable vertical extent of the spine, and is specially intense in certain spots, usually in more than one. The dorsal region is the most common seat, next the lower cervical, and least frequently the lumbar region. Sometimes the pain is localised on one side of the spine, close to it. That which is felt in the spine in cases of gastric ulcer seems to be a sort of reflex neuralgia. Often the pain seems to pass up to the back of the head. Spinal neuralgia may be associated with a similar pain in some other part of the trunk, in the shoulder, arm, or leg. It is usually accompanied by considerable tenderness, and is increased by fatigue, by use of the legs, by long sitting or standing, and also by vibration, such as the movement of a carriage. The latter point is often of considerable diagnostic importance, for it is far more marked in neuralgia than in spinal diseases causing pain. On the other hand, the pain is not increased by slight movement, as is the pain of growths and caries. It is seldom distinctly paroxysmal, but, as already mentioned, in one case paroxysms of pain in the cervical spine and vertex were most intense, and each was accompanied by opisthotonos. It is uncertain in what structure this spinal neuralgia is produced. It is often associated with neuralgia elsewhere, and also with rheumatism of the fibrous tissues, so that some cases seem to be of the nature of rheumatic neuralgia. It has been thought that the membranes are the seat of the pain, but there is no real evidence for or against the theory.

Another very common seat of neuralgia is the *sacral region*, no doubt in consequence of the plexus of nerves that lies between the bone and the skin. Pains of pelvic origin are often referred to this region, as those of parturition show. Occasionally the pain is felt chiefly about the coccyx. It must be remembered that the fibrous tissues over the sacrum are sometimes the seat of acute rheumatic pain, such as, higher up, is termed lumbago.

Neuralgia of the Leg.—In the lower limb, neuralgia is rare in the region supplied from the lumbar plexus, although a *crural form*, in which the pain occupies chiefly the front of the thigh, is occasionally met with. In most instances pain in this region is of secondary origin, due to a lesion of the lumbar plexus, as, for instance, from the pressure of an abdominal tumour, or is due to the extension of neuritis from the sciatic up the lumbo-sacral cord.

The majority of neuralgic pains in the leg are in the region of the sciatic nerve, and are grouped under the designation of sciatica. The pain occupies especially the course of the nerve, but tender foci are met with in certain spots;—*lumbar*, near the spine, just above the sacrum; *sacro-iliac*, at the articulation of the same name; a *gluteal*, opposite the middle of the lower border of the gluteus; a series of spots, varying in exact position, along the course of the nerve in the posterior region of the thigh; a *peroneal* behind the head of the fibula; a *malleolar* behind the lower extremity of the fibula, and an *external plantar* at the outer border of the foot. It is, however, certain that sciatica is seldom a true neuralgia. Almost all severe cases are due to inflammation of the nerve-trunk; the evidence of this has been stated in the account of the disease in the first volume.

Lastly, it should be mentioned that, in rare cases, neuralgic pains are felt almost everywhere, in the limbs, trunk, and head, and apparently constitute a sort of universal neuralgia. The few cases I have seen have been in adult men, and associated with hypochondriasis.

Varieties depending on Character and Cause.—Epileptiform Neuralgia.—The term is applied (according to the example of Trousseau) to an intractable form, in which each attack of pain comes on very suddenly, with intense severity, and lasts usually less than a minute, sometimes only a few seconds, rarely two or three minutes. The frequency with which the attacks recur varies in different cases; the daily number may be one, a hundred or more. The pain is most intense, so that the patient stamps about the room in agony, or tries to get relief by violent rubbing or by pressure. Trousseau relates the case of a lady whose malar bone was atrophied in consequence of the pressure, repeated every few minutes for years. The sufferers from this terrible form are usually in the second half of life. The pain is invariably in the region of the fifth nerve, sometimes in the whole, sometimes in part only, but rarely confined to a single branch. Convulsive spasm in the face may accompany the pain (convulsive epileptiform neuralgia).

Reflex or sympathetic neuralgias are those in which the pain is felt in another nerve-region than that in which its cause exists. To radiation of neuralgic pain must, as already stated, be ascribed to the extension of the central discharge to adjacent centres, but the term "reflex neuralgia" is not applied to such cases, but to those either in which the pain exists only at a distance from its cause, or in which the distant neuralgia has no sensory continuity with the primary pain. The pain of a carious tooth may be associated with a distant and apparently disconnected pain in some other part of the fifth nerve, or such disease may cause only distant neuralgia, and no local pain. The proneness of the fifth nerve to be affected in neuralgia renders it a common seat of the reflex form. For instance, neuralgia limited to the fifth has been produced by injury to the ulnar nerve, and also to the occipital nerve (Anstie). Nevertheless, the converse relation is sometimes observed; carious teeth are said occasionally to cause neuralgia in other parts, as, for instance, in the cervico-brachial region (Salter). When no local pain is felt, we must assume that the centre to which the afferent impressions directly come, is not thus excitable to painful activity, or even so as to influence consciousness, but that it is in connection with another centre, which, by natural or acquired susceptibility, is disposed to excessive action. Curious cases are on record in which the act of micturition invariably caused a pain in the region of the arm supplied by the ulnar nerve.*

The cause and seat of reflex neuralgia may be most varied. When due to causes acting on the cerebro-spinal nerves, the pain is usually felt on the same side, and rarely far distant. A most important class of reflex pains are those which are produced by disease of the internal viscera, such as the spinal pain in ulcer of the stomach, the sacral pain in uterine disease, the scapular pain in affections of the liver, the pain in the testicle in renal colic, and pain in the front of the thigh and spine in gonorrhœal orchitis (Mauriac). Many of these are rather reflected pain than neuralgia properly so called, but they occasionally persist when their cause is removed, or assume disproportionate intensity. They are of very great practical importance on account of the readiness with which their cause may escape attention.

Traumatic neuralgias are those that are excited by injury to nerves —by contusion, and by punctured, lacerated, or incised wounds. In some instances the pain is the result of a neuritis, set up by the injury, which may ascend the nerves and pass, at a junction, to other trunks. But neuralgia may also be set up when there is no more than the local and transient inflammation at the spot injured. Such neuralgias may follow nerve-lesions in any part of the body, but are especially frequent in the arm, the nerves of which are much exposed to injury. In the days of venesection, a common form of neuralgia resulted from injury to a cutaneous nerve in the operation. It may follow wounds of both large and small nerves, and by some observers has been thought to be more frequently due to the latter, but it must be remembered that small nerves are more often wounded than larger trunks. Anstie believes that neuralgia is more common from partial injuries than when the nerves are completely divided. It is a frequent consequence of gunshot injuries of nerves. In some cases the disease is apparently due to the implication of nerves in a cicatrix, and the morbid enlargement of the nerve-ends after amputation, which goes by the name of "bulbous nerves" and may cause much suffering.

The pain may commence a few hours or days after the injury, but more frequently not until after an interval of some weeks or months. It generally begins in the point injured, but is rarely limited to this, or even to the part supplied by the wounded branch. It usually radiates to adjacent regions, and sometimes to parts supplied by other nerve-trunks. For instance, a woman received a blow on the thorax, probably contusing an intercostal nerve. The early pain of the injury ceased, but two months later, neuralgic pains commenced at the spot, and gradually spread over the whole region supplied by the cervical and brachial plexus (Ollivier). The pain may be less intense at the seat of the injury than it is elsewhere, and it may even be absent at the former, and then is of the purely reflex variety, instances of which have been already mentioned. These facts, and also the circumstance that the pain is not always influenced by excision of the injured nerve, show clearly that it is to a large extent of central origin, the expression of a disturbance which, though excited by the nerve injury, is to a considerable extent independent of it, and doubtless due, not only to the excitant, but also to a predisposition, such as is concerned in the production of other neuralgias. The same fact is seen even more clearly in cases in which some injury leaves a slight local weakness or disability, and, years afterwards, the part may become the seat of neuralgic pain, under the influence of a constitutional tendency.

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* Several such cases are given by Vanlair ('Les Nevralgies,' p. 330); one of them comes from a curious source,—the autobiography of Clarendon, Chancellor to Charles II. See note on p. 799.

The pain is usually intermitting in character, but often most intense in degree, and causes profound depression of the general system. It may excite muscular spasm, and also, especially soon after the injury, other reflex symptoms such as vomiting. Vaso-motor disturbance occasionally ensues; trophic changes in the skin and joints have been described, but it is very doubtful whether nutrition suffers except in consequence of considerable secondary neuritis. These lesions are considered more fully in the chapter on neuritis in the first volume. The course of traumatic neuralgia is often tedious, sometimes most prolonged, and it has been known to last to the end of life, in spite of every medical and surgical measure that could be devised for its relief. When it ceases, it is prone to recur under the influence of general depressing influences.

Occupation-Neuralgias.—These are pains, more or less neuralgic in character, brought on by some habitual act. They are the sensory forms of occupation-neurosis, in the account of which they have been fully described.

Herpetic Neuralgia.—Herpes zoster, whatever its seat, is usually accompanied by pain of a neuralgic character. The explanation of this is found in the conclusive evidence that the eruption is the effect of nerve irritation, probably always inflammatory in character. It corresponds in area to the distribution of certain nerves, and, post mortem, inflammation has been found in the nerve (Haight) and in the ganglia of the posterior roots (Bärensprung, Charcot, &c.). The neuralgic pain is thus "symptomatic," the result of organic change in the nerves. Sometimes herpes occurs in the area of a nerve which has long been the seat of neuralgic pain, and hence is regarded as an occasional trophic effect of neuralgia, but it is an extremely rare consequence, and probably only occurs in cases in which the pain is the expression of organic changes, and is not a consequence of simple idiopathic neuralgia.

In cases of ordinary zoster, pain may occur before or after the eruption. The initial pain precedes the appearance of the eruption for a few hours or days, is usually moderate in severity, and commonly (but not always) subsides as the cutaneous lesion is developed. Sometimes this pre-herpetic pain is absent, especially in the young, although even then there is usually an initial sensation of tingling in the part. The pain that succeeds herpes is more constant. It comes on usually during the decline of the eruption, and its occurrence bears no relation to the seat of the herpes. The sequel is most frequent where herpes is most common,—the side of the trunk, the forehead, neck, and leg. The pain has the distribution of the eruption, corresponding to the nerve or nerves affected. It varies in degree, but is usually acute, lancinating, and for a time severe, and is accompanied by great tenderness of the skin. The same tender points are met with as in ordinary neuralgia in the same area. There is an important relation between the age of the patient and the severity and duration of the pain. In old persons it constitutes one of the most severe and persistent forms of nerve pain, often continuing for months, and even for years, before, at last, it slowly lessens. Occasionally it persists in unmitigated intensity to the end of life. Sir William Jenner, in his lectures, was accustomed to illustrate the obstinate persistence of this pain in the old, by the instance of a man who, before the days of anæsthetics, endured the excision of the skin to which the pain was referred, in the hope of relief, but found none, and then, unable to bear the continuous agony, he shot himself.

Hysterical Neuralgias.—Neuralgic pains are common in hysteria, but a distinction must be drawn between those which are merely associated with, and those which are due to, the general neurosis. Of associated forms, every variety of true neuralgia may be met with, due to the neuropathic disposition, which is also the cause of the hysteria, and they present the characteristic distribution, tender points, &c., of the ordinary form. Anæmic neuralgias are also common in hysterical patients, in whom the nerve disturbance is often due to poorness of blood. Of the neuralgic pains that are due to hysteria, some are in the cerebro-spinal system, some in that of the sympathetic. Certain local pains in the head are very common, but these differ from ordinary neuralgia in being confined to one small spot, instead of following the course of nerves, and the local pain has a sharp stabbing or boring character, as if a nail were being driven in, and hence has received the name of "clavus hystericus." It is important to remember that a similar pain may be met with apart from hysteria, especially in anæmic persons. Pains in the spine are also extremely common, sometimes very local, and of various characters. Other pains referred to the trunk are usually associated with local tenderness in the so-called "hysterogenic points." The most important neuralgias of hysteria are those of the viscera, ovarialgia (not necessarily in the ovary itself) and gastralgia being the most frequent. Pains in the joints and muscles are also common. The visceral neuralgias, and those associated with parietal tenderness, are usually persistent, but the local boring pains, and those in the joints and muscles, are often fugacious and migratory, and this constitutes a diagnostic point of considerable importance.

Rheumatic Neuralgia.—In a loose way, all neuralgias produced by cold are sometimes styled "rheumatic," but the mere causal relation scarcely warrants the epithet. The peculiar affection termed "muscular rheumatism" is also sometimes called a rheumatic neuralgia, but this is to extend the use of the term in a manner that is scarcely justified or needed. Pain that occurs only on movement should never be called neuralgia. Acute articular rheumatism is rarely associated with true neuralgia, but some forms of spontaneous pain are frequently produced by cold in those who present what is termed the "rheumatic diathesis," who perspire easily, are liable to catarrh, and whose urine readily becomes loaded with lithates after a chill. Such pains may correspond to a certain nerve, or may occupy some part of a limb, without any definite relation to nerves, sometimes with a definite relation to the muscles or fibrous tracts, and are often migratory. Their exact pathology is uncertain. Some other facts regarding this variety have been mentioned on p. 796.

Gouty Neuralgia.—The subjects of gout not unfrequently suffer from nerve-pains, apparently idiopathic in character, coming and going, and sometimes very severe. The pain may disappear when an attack of acute gout is developed. The fifth, the intercostal, and the sciatic nerves are those most frequently affected. Severe sciatica sometimes occurs in the gouty, but is certainly due to neuritis. One visceral neuralgia is also sometimes due to gout, gastralgia.

Diabetic Neuralgia.—Patients with diabetes may suffer from neuralgic pains that have no special characters, but Worms has called attention to the symmetry of the pains, which occupy the same nerve on both sides, as a characteristic of diabetic neuralgia. The pain has hitherto been observed chiefly in the third division of the fifth nerves and the sciatics. It must be remembered, however, that ordinary neuralgia is occasionally symmetrical. The nerve-pains met with in diabetes are often severe and obstinate until the cause is removed by dietetic treatment. They have been observed to increase and decrease with the amount of sugar in the urine. They are sometimes the result of a peripheral neuritis.

Anæmic Neuralgia.—Anæmia is one of the most powerful causes of neuralgia in all its forms, but certain varieties are more frequent than others in this condition, especially in young women. One of these is situated in the fifth nerve, either in the first division or in the auriculo-temporal branch. It is generally intermittent and is increased by movement, while it is lessened by the recumbent posture. Another, still more frequent and more continuous, is that which is felt in the thorax, in the fifth and sixth interspaces on the left side, the well-known infra-mammary pain. Gastralgia is also common apart from ulcer. Headaches that have no true neuralgic character are also very common.

Neuralgia of the cerebro-spinal nerves sometimes occurs in *lead-poisoning*, but it is often uncertain whether the pain is the expression of functional disturbance or is due to neuritis. The knowledge of this cause, however, is very important. It has been thought that lead-colic is in part a neuralgia; the evidence of this is insufficient, but there may be severe epigastric pain.

Malarial neuralgias are not very common even where ague is frequent. They present nothing characteristic in seat, although the supra-orbital and intercostal forms are the most frequent; nor is there anything special in the character of the pain. Their chief feature is regular periodicity, the intervals between the attacks being from one to four days. The periodicity is less characteristic when the attacks are diurnal than when three or four days intervene. Occasionally the paroxysms are attended with slight symptoms of an ague fit, a trifling cold and hot stage (Anstie). These must not be confounded with the vaso-motor phenomena met with in cases of the ordinary form. It is very doubtful whether the neuralgia is, in most cases, a direct effect of the malarial poison, in the sense in which ague is. It is probably an indirect effect, the result of the anæmic and depressed state of the nervous system induced by malarial influences, even in those who do not suffer from intermittent fever. Neuralgia, apparently due to malarial causes, does not always yield to quinine, even when given in the most approved manner. It is probable also that the periodicity of many malarial neuralgias is the result of the state of the nervous system produced by the poison, and it does not prove the neuralgia to be truly malarial. In some cases of supra-orbital neuralgia with exact periodicity, coming from malarial districts (recorded by Seeligmüller), quinine failed entirely while other treatment was quickly successful.

Syphilitic Neuralgia.—The pains of syphilis constitute a prominent symptom of that disease, but, for the most part, have no correspondence with nerve distribution, and can therefore be scarcely regarded as neuralgic. Symptomatic neuralgic pain occurs in many syphilitic affections of the nervous system which cause irritation of the nerves or their roots, in chronic meningitis, neuritis, and pressure from growths. One of the most severe and obstinate cases of pain in the region of the fifth nerve I have seen, was due to chronic syphilitic meningitis at the origin of the nerve. In such cases the nature of the lesion is usually clear from the evidence of structural damage to the nerve-fibres. In the case just mentioned, for instance, there was anæsthesia and paralysis of the masseter. Whether idiopathic neuralgia results from the influence of the syphilitic poison is uncertain. Fournier believes that such neuralgia is common during the secondary stage, but very few conclusive cases have been recorded. It must be remembered that in the early stage of neuritis, &c., pain may be the only symptom, and, on the other hand, the anæmia which results from syphilis may be the real cause of the neuralgia. Anstie believed that syphilis does not cause true neuralgia although it may recall a neuralgia which had long ceased. A peculiarly distressing post-sternal pain, apparently neuralgic in character, has been occasionally observed in constitutional syphilis (Eccheverria, Buzzard).

Degenerative Neuralgia.—In advanced life, and sometimes before the senile period is reached, neuralgia is occasionally met with, of extreme obstinacy, and associated with other signs of degeneration of the central nervous system, such as failure of memory or persistent mental depression. The neuralgia is apparently one effect of the degenerative tendency. The affection has all the characters of a central neuralgia. The fifth nerve is by far the most common seat, but the pain sometimes occurs in other situations.

DIAGNOSIS.—The diagnosis of neuralgia rests on the unilateral situation of the pain, on its correspondence to the distribution of certain nerves, its intermitting or remitting character (*i. e.* the occurrence of paroxysmal exacerbations), on the fact that the patient has suffered from similar pains elsewhere, on the variations in the seat of the pain, and on the absence of any indications of actual damage to the nerve-fibres. The variability is a symptom of great importance. If the pain shifts about, now in one spot and now in another, it is not likely to be due to an organic cause. For instance, a man with fronto-occipital neuralgia had foci of pain in the forehead, temple, and occiput, but he never had pain at the same time at more than one of these places. The last of the above indications, however, is the most important element in the distinction of neuralgia from the similar pains which result from organic disease of the nerves due to external pressure or neuritis. It is in the case of the fixed neuralgias (as distinguished from the migratory forms) that the distinction is of chief importance. The diagnosis is more difficult in the case of neuritis than of external pressure (tumour, &c.), because, in the latter case, the cause of the pressure usually produces other symptoms, and the effect of the pressure is progressive, so that gradually increasing signs of a structural lesion are added to the pain. But from neuritis the distinction may be much less easy. The difficulty is greater, the slighter the degree of neuritis. Severe inflammation causes severe, constant pain, at first more intense at its seat than in the distribution of the nerve, because the inflammation is most intense in the nerve-sheath, and the sheath-nerves suffer first. In severe forms, however, the proper fibres of the nerve are soon implicated to a degree that interferes with their conducting functions, so as to cause, first, hyperæsthesia, and afterwards areas of anæsthesia, while if the nerve is "mixed," the muscles supplied become weak, flabby, and rapidly waste, with changes in electrical irritability. In slight cases, on the other hand, the sheath chiefly suffers; there may be no interference with conduction, and the pain resembles neuralgia more closely, although, as a rule, it is more continuous than in true neuralgia. The diagnosis is also difficult when the seat of the neuritis is such as to render the nerve inaccessible to direct examination. If it can be reached, there will be found, from the first, local tenderness of the nerve. In idiopathic neuralgia, tenderness of the trunk in the intervals is only developed after the neuralgia has existed for some weeks. Moreover, in neuritis, distinct swelling of the nerve can sometimes be felt. Local tenderness is thus chiefly of significance in the early stage of the affection, or when it occupies a considerable area of the nerve-trunk, and is not confined to certain points.

If, therefore, the pain is migratory, if it is completely intermittent, especially if the intermissions are of long duration, if the attacks are induced by psychical influences, the suspicion of an organic cause will scarcely arise. If, on the other hand, there is continuous pain, rapidly developing to a considerable degree, organic disease should be suspected, and the suspicion will be confirmed if there is evidence of early or considerable impairment of the conducting function of the fibres, persistent alteration of sensibility, muscular wasting or altered irritability, or trophic changes in the skin. Great care is necessary, however, in testing the muscles, because a strong electrical stimulus may greatly increase the pain. The isolated faradic shocks should be employed rather than the current, because they cause much less pain, and because they detect most readily the earliest change produced by neuritis, a slight *increase* of irritability; an altered reaction to voltaism will also often be found. Early tenderness of the nerve, not merely during, but between the paroxysms, indicates neuritis, but its absence does not exclude inflammation, which may occupy an inaccessible portion of the nerve. If the symptoms of structural damage gradually increase and progress, and especially if they involve the whole region of the distribution of the nerve, compression may be suspected, and is confirmed by the discovery of any other symptoms indicating organic disease in the vicinity of the nerve-trunk, such as, in the case of the fifth, damage to other nerves which arise or pass near it. It must be remembered that in some cases the differential diagnosis of slight inaccessible neuritis from neuralgia may be very difficult, because, on the one hand, the irritation of neuritis may cause neuralgic changes in the nerve-centre, and, on the other, a primary functional neuralgia may cause, by reflex vaso-motor disturbance, secondary changes in the sheath, so that in each case a mixed affection, partly functional, partly organic, is the result; or, in current terms, a neuralgia which is at first either symptomatic or idiopathic may ultimately be both. At the same time, the diseases are often confounded through simple ignorance or misconception. The latter cause, as we have seen, often leads sciatic neuritis to be regarded as neuralgia; and the former, with remarkable constancy, causes brachial neuritis, severe and characteristic, to be thought a mere neuralgia.

In all forms the circumstance that the patient has previously had attacks of neuralgia in other situations is a very important help in diagnosis. It does not, of course, prove that a given pain is of functional origin, but it is proof of a tendency to such pain, which may justly be allowed considerable weight in the absence of signs of an organic lesion. This character is frequently of very great practical value, especially in cases of neuralgic pain of unusual seat. Another similar indication is furnished when the neuralgia replaces some other functional affection, as, for instance, migraine or melancholia.

Special Diagnosis.—There are certain affections with which the several varieties of neuralgia are liable to be confounded. Almost any form of neuralgia may be simulated by the pains of tabes, and the possibility of this cause should always be thought of, especially in the case of migratory pain. In some situations the risk of error is greater than in others, and these will be especially mentioned.

In neuralgia of the nerves which are distributed over the skull (fifth and great occipital) it is often doubtful whether the affection should be called headache or neuralgia. The former may be local, *e.g.* at one temple, as in migraine. The chief distinction is the correspondence to the course of the nerves, or to an entire nerve-region, in neuralgia, which is absent in headache. But true neuralgia may induce a diffuse head-pain which may be called "neuralgic headache." The fifth nerve is often damaged by organic intra-cranial disease, and nerve-pain, thus produced, is sometimes mistaken for simple neuralgia. Besides the indications already described, organic disease often causes deeply seated headache and various symptoms, especially in the functions of other nerves, optic neuritis, and paralysis or convulsion in the limbs. The occurrence of herpes in the course of a supposed neuralgia is also evidence of organic change in the nerve. It must not be forgotten that neuralgic pains are sometimes the first symptom of morbid growths in the upper jaw and parotid region.

The diagnosis of the cervico-occipital form rarely presents any difficulty. The neuralgic pain usually courses along the nerve-trunks, but it must be remembered that occipital neuralgia is occasionally bilateral. In caries of the cervical vertebræ, pain may be an early symptom, but it scarcely ever spreads to the occiput, and the early interference with movement is usually characteristic.

The neuralgias of the arm have to be distinguished chiefly from neuritis by the indications already mentioned. The diagnosis is seldom difficult, because neuritis usually causes early tenderness in the affected nerve-trunks, and, later, trophic changes in the muscles and skin.

A more difficult problem is presented by the trunk neuralgias, which have to be distinguished from disease of the internal viscera, and from organic spinal disease. Unilateral pain is a frequent accompaniment of disease of the organs in the thorax and abdomen, and it is hardly necessary to point out that, in every case, a careful examination should be made of the organs adjacent to the seat of pain. The greatest difficulty arises in the case of deeply seated tumours which cause pain by nerve-compression, especially when, as is sometimes the case, the pains are "reflected," and do not precisely correspond in position to their cause. Aneurism of the aorta, for example, often gives rise to such pain, especially when seated in the descending part; pain in the course of the nerves may be the only symptom until sudden death occurs. The pain is usually very severe, and often burning in character, and has not the same foci of intensity and tender points as ordinary neuralgia. In severe unilateral (and even bilateral) pain, persistent in occurrence, whether uniform in seat or not, this cause should always be suspected. One of the most severe cases of neuralgic pain I have ever seen—darting, stabbing, burning, migratory pains in legs, abdomen, thorax, and left arm—was due to an undiscovered abdominal aneurism. Aneurism of the ascending part of the arch is now and then accompanied by pain passing to the arm, apparently of reflex character. Intercosto-humeral neuralgia may simulate angina pectoris, of which, indeed, such neuralgia may be said to form part. The severity of the paroxysms of angina, and the other distressing sensations which accompany the pain, usually render the nature of the attacks sufficiently clear.

The parietal pains which accompany disease of the spinal cord rarely have the acute lancinating character of neuralgia: they are sensations of tightness or constriction, and the obtrusive symptoms in the legs indicate the nature of the disease. To this, however, morbid growths in the membranes offer an exception, especially in the early stage. The pains may be very severe and of various character, but they are constant in seat, are increased by any movement, and, before long, indications of compression of the cord are added to them. The "lightning pains" of tabes are sometimes felt in the trunk, and have often been mistaken for neuralgia. They are distinguished by their changing seat and momentary duration, by the similar pains in the legs, and especially by diminished sensibility over extensive areas. In most cases there are some pains in the legs and the knee-jerk is lost; I have seen one case in which the changes were confined to the dorsal region of the cord, and the knee-jerk was normal; but in this, as in other cases of the kind, the light-reflex of the iris was lost—a symptom of great indirect diagnostic importance. More constant in seat and neuralgic in character are the pains of pachymeningitis, which are caused by the compression of the nerve-roots by the thickened membrane. They are distinguished from neuralgia by the wide extent of the pains, their bilateral situation, by the presence of areas of anæsthesia due to still greater damage to the nerves, by muscular wasting in the limbs, and by the symptoms of compression of the cord.

The terrible nerve-pain which is sometimes produced by organic disease of the bone of the spinal column closely resembles neuralgia in its fixity of site and unilateral situation, but is distinguished by its peculiar dependence on movement of the trunk (see vol. i, p. 257). Leg symptoms are usually soon associated with it. The lumbo-abdominal neuralgia may be confounded with renal colic. The distinction rests chiefly on the urinary symptoms that accompany the latter.

The sharp pains of tabes are more frequently felt in the legs than in the trunk, and there also are often mistaken for neuralgia, but the indications already mentioned suffice to distinguish them. Occasionally such pains are met with apart from other symptoms of tabes, and their nature is difficult to determine. If they are the sequel of syphilis, or associated with loss of action in the iris, they are probably the evidence of a partial tabetic degeneration—a tabetic neuralgia. The distinction of sciatic neuralgia from neuritis must be made by the indications already given (see also "Sciatica," vol. i, p. 106). Of still greater practical importance is the distinction of crural and sciatic neuralgia from the pains due to pressure on the lumbar and sacral plexus by tumours in the pelvis and abdomen. Such pains are felt along the course of the nerves, and are almost invariably thought at first to be neuralgic. Pains in the front of the thigh are rare except as the result of extension of neuritis from the sciatic nerve to the lumbar plexus, or as the result of pressure. In each case there is generally muscular wasting, which shows organic damage. The diagnosis between neuralgia in the front of the thigh and pain due to a lesion of the nerves, is also aided by the state of the knee-jerk, which is generally early lost in organic disease, but remains intact in neuralgia. In every case of neuralgic pain in this situation, the abdomen should be carefully examined, and whenever pain in the sciatic is of a progressive character, and apparently due to mischief above the accessible part of the nerve, a rectal examination should be made, by which the source of pressure, if there be any, will often be felt. It must also be remembered that pain felt in the knee may be a reflex effect of the irritation of the branches of the obturator nerve in hip-joint disease, and that obscure pains in the groin and thighs are sometimes the result of disease of the femur.*

The diagnosis of the special forms of neuralgia is less difficult. In the reflex or sympathetic variety the cause of the pain will usually be discovered if it is remembered that it may be outside the area in which pain is felt. It is rarely far distant; often in another branch of the same nerve. Pain in any part of the fifth nerve, for instance, may be due to the irritation of a carious tooth.

It is possible to confound epileptiform neuralgia with the form of true epilepsy in which the aura is a sudden pain. I have known, for example, slight attacks of minor epilepsy to be preceded by a most severe momentary pain in one fifth nerve. The occurrence of distinct loss of consciousness, and still more of convulsion, sufficiently indicates the epileptic nature of the case. A painful epileptic aura in a limb, followed by local convulsion, could only be mistaken for neuralgia with reflex spasm in a patient who had never had a severe fit. But the course of such an aura is usually centripetal and deliberate, and the spasm has also a deliberate march.

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* In one case of intense stabbing pains in the groin, closely resembling neuralgia, the cause was ultimately found to be necrosis of the great trochanter.

PROGNOSIS.—The prognosis in neuralgia is influenced by the age of the patient, by the duration of the affection, its situation, severity, and cause. It is far better when the disease is due to any constitutional condition than when no general cause can be discovered, since, as a rule, the constitutional states that cause neuralgia are amenable to treatment, at any rate in such a degree as to influence the pain. In hysteria, however, some neuralgias are readily removed; others, especially when there is no anæmia, are most obstinate. The prognosis is better when there is no hereditary tendency than when this is marked. We do not yet know whether the prognosis is influenced by the fact that the heredity is general or special. The more severe the pain, and the longer the disease has lasted, the more difficult is its treatment. When a patient has suffered from neuralgia in various regions, for many years, the ultimate prognosis is less favorable. It is generally believed that neuralgias of the fifth nerve are more intractable than others. During the decline of life, most varieties, especially those of the degenerative form, are peculiarly obstinate, and in old age they sometimes cannot be relieved by any treatment whatever. However severe the pain may be, neuralgia involves little danger to life; as Buzzard has well said, "the disease does not seem, of itself, to shorten the duration of the life which it fills with suffering." Epileptiform neuralgia is, of all kinds, the most obstinate; Trousseau, in his large experience, never knew a case to be cured. When neuralgia has once ceased it is extremely prone to recur; and this fact, which is true of all varieties, must be remembered in giving an opinion regarding the future.

TREATMENT.—The treatment of neuralgia consists, first, in the relief of the symptom, pain; and secondly, in the removal of its cause, *i. e.* in the restoration of normal conditions of function in the sensory apparatus. The means by which these ends are to be secured are threefold,—by hygiene, by drugs, given internally and applied externally, and by certain surgical operations on the nerves. It is evident that the treatment to be adopted must be influenced, to some extent, by the nature of the case. When there is distinct evidence of neuritis, the treatment for this, already described, must be adopted.

That which has now to be considered is the treatment of the "idiopathic" form, but the means for the alleviation of all varieties is nearly the same.

Although the relief of pain has usually to be the first actual step in treatment, the first in importance is the removal of the causes of the disease. Any discoverable condition on which the neuralgia may depend must be treated. The detailed measures that are necessary need not here be indicated, since they are sufficiently suggested by the enumeration of the causes and causal varieties already given. Especially should any source of nerve irritation be removed, whether in the region of the painful nerve or outside it. It must be remembered, however, that it is not certain that the neuralgia will disappear on the removal of such a source of irritation. This is often true of neuralgias of the fifth nerve and decayed teeth. The almost universal association of neuralgia with conditions of debility, so strongly insisted on by the late Dr. Anstie, indicates the importance of hygienic measures calculated to improve the general health; fresh air, adequate rest, and nutritious food in full quantity, with a small quantity of alcohol at meals, are usually directly beneficial. The importance of a good supply of animal food is of great importance for all but gouty subjects. I have known severe neuralgia to occur first on the patient commencing a purely vegetable diet, to disappear when meat was taken, and recur with severity at each of four successive attempts to return to vegetarianism. Cod-liver oil, or other easily digested fat, may often be added with advantage. Iron, when there is anæmia, will sometimes alone cure the disease, and even when there is not anæmia it seems occasionally to be beneficial. Of nervine tonics, quinine is of most value in true malarial forms, given in a full dose shortly before a periodical attack is due; but, as we have seen, it is not always successful. In smaller doses it is occasionally useful in other forms, although not so frequently as might be expected from its marked influence on the nervous system. In stupefying doses it may lessen the pain for a time, but does not often produce a lasting effect. It has been thought to be most useful in neuralgias of the first division of the fifth nerve. Nux vomica or strychnine often does more good than quinine. In general it may be said that whenever the nervous system is feeble, as it so frequently is in neuralgia, nux vomica may with advantage be added to other remedies that are given. Zinc (including the phosphide) is of little value. Arsenic is occasionally useful, especially in the neuralgias that have been set up by malarial poisoning, and in the degenerative variety. Free phosphorus ($\frac{1}{50}$ or $\frac{1}{30}$ grain) has been strongly recommended, but my experience coincides with that of most recent writers in assigning it a very low position in the list of remedies,—low so far as numerical success is concerned, although in rare cases its influence is very striking. For instance, a woman aged forty-three, with neuralgia of the fifth nerve of thirteen years' duration, at one time associated with brachial, and afterwards with crural, neuralgia, lost the pain entirely during three months' treatment with phosphorus, although when the treatment was commenced about twelve severe paroxysms occurred every day. Ammonio-sulphate of copper has been recommended in cases of neuralgia of the fifth nerve. It may be given in doses of $\frac{1}{16}$ or $\frac{1}{12}$ of a grain after food. In syphilitic forms, dependent on actual nerve-lesions, iodide of potassium and mercury should of course be given. Iodide of potassium is now and then useful in cases of neuralgia that are not due to syphilis.

Of remedies that have a sedative action on the nervous system, although they are not anodynes, bromide of potassium has been most used. It is occasionally of service in cases of idiopathic neuralgia, paroxysmal in occurrence, although it appears to have less influence on sensory than on motor nerve-cells. Its value is greatest in irritable, anxious subjects. Chloride of ammonium has been recommended in intercostal neuralgia. In the rheumatic forms salicylate of soda has been occasionally found useful, especially in facial neuralgia and sciatica, but in the cases of sciatica which depend on neuritis it has little influence. In some cases, marked relief is afforded by antipyrine, acetanilide (antifebrin), exalgin, and phenacetin; they are somewhat uncertain in their influence, which is sometimes great at first and afterwards slight.

Nervine stimulants, given at the beginning of an attack, occasionally cut it short, especially in neuralgias of the fifth nerve. Sulphuric ether, valerian, and alcohol have been employed for this purpose. The use of alcohol is, however, fraught with great danger to the habits of the patient, and should be avoided as far as possible. Another nervine stimulant, turpentine, is an old remedy for sciatica, and has been occasionally of service in cases of ordinary neuralgia. Nitroglycerine sometimes gives relief to the pain, and, as a rule, it succeeds whenever alcohol is effective. It is also extremely serviceable in many cases, given regularly three times a day, in doses from $\frac{1}{200}$ to $\frac{1}{50}$ of a grain, especially in combination with nervine tonics; the dilatation of the arteries seems to favour the access of the tonic to the nerve-tissues.

Anodynes are necessarily very important agents in the treatment of the disease. It is the pain for which the patient seeks help; the cause of the pain can only be slowly influenced, and in the meantime the pain itself has to be relieved. In some cases relief is all that can be afforded; treatment fails to prevent the recurrence of pain, and all that can be done is to lessen its intensity. But in a large number of cases of neuralgia, anodynes do more than merely palliate; the repeated removal of the pain tends to prevent its recurrence. This makes it probable that the relief afforded is not merely by an action of the drug on the general sensorium, but that it has an influence on the specific disturbance which gives rise to the sensation of pain. The same conclusion is suggested by the fact that the most acute pain may be sometimes relieved by an anodyne which has no apparent influence on the general sensory functions of the brain.

No drug gives relief so quickly and so surely as opium or morphia. In epileptiform neuralgia Trousseau found no remedy comparable to opium, which he gave in doses rapidly increased up to 300 grains of opium or 60 grains of sulphate of morphia a day. Morphia beneath the skin relieves for the time all forms of neuralgia. It is probable that, in most cases, it is better to inject it into the seat of pain, because it can then exert some influence on the terminal nerve-endings, and, moreover, the injection has a slight counter-irritant influence. But its chief action is on the nerve-centres, and this is exerted equally wherever the injection is made; therefore, if for some reason, local injection is undesirable, any convenient region will answer. The dose should not at first be large, $\frac{1}{10}$, $\frac{1}{8}$, or $\frac{1}{6}$ of a grain, according to the severity of the pain. It is unsafe to commence with a larger quantity; death has, in more than one instance, resulted from an injection of $\frac{1}{4}$ gr. It is remarkable, however, in some cases, how little effect, beyond the relief of the pain, morphia produces. The addition of atropine (in the proportion of one-twentieth of the morphia used) will often prevent nausea. Caution should be used to prevent the dangerous "morphinism," most cases of which have been set up by the use of this drug for the relief of neuralgia. The hypodermic syringe should never be placed in the hands of the patient. In cocaine, moreover, we have an agent that can often replace morphia. A local injection of from half a grain to a grain often gives great relief to the pain, apparently by arresting impressions from the periphery, but the first injection may wisely be smaller, $\frac{1}{12}$ gr., on account of individual intolerance. Repeated injections often promote recovery, especially when the pain is superficial and is accompanied by tenderness.

Belladonna may be given by the mouth ($\frac{1}{8}$—$\frac{1}{6}$ gr. of the extract) or atropine by the skin ($\frac{1}{120}$—$\frac{1}{60}$ gr.). Occasionally these give marked relief to the pain, and when this is the case they are said to produce a more permanent effect than morphia (Hunter, Anstie, Vanlair) The unpleasant dryness of the throat cannot be avoided. It is often well to alternate injections of atropine with those of morphia in cases in which there is danger of the establishment of the morphia habit, and cocaine is insufficient.

Aconite and gelsemium are said to have a special action on the fifth nerve, and are frequently useful in trigeminal neuralgias. Aconite is the more powerful of the two, but often causes nausea and unpleasant symptoms. In slight cases, especially those which depend on dental caries, gelsemium is sometimes useful; fifteen minims of the tincture may be frequently repeated. Aconitia may also be given in doses of $\frac{1}{250}$ to $\frac{1}{100}$ of a grain, but it is a somewhat dangerous remedy to employ hypodermically. The alkaloid of gelsemium (gelsemia) is convenient for subcutaneous use; the dose is $\frac{1}{60}$ to $\frac{1}{30}$

of a grain. Cimicifuga is often very useful for neuralgia associated with rheumatism, given either alone or combined with Indian hemp. In gouty subjects, lithia may be combined with the cimicifuga.

Indian hemp is of value in certain forms of neuralgia, especially those in which the pains are sudden, brief, and sharp, without the violence which marks true epileptiform neuralgia. It may be administered continuously between the intervals; a quarter to half or even one grain three times a day. Piscidia erythrina has been recommended when pain is not severe; in moderate doses (ʒss of the liquid extract) it leaves no unpleasant after-effects. Chloral has little influence over pain, but croton-chloral (butyl-chloral) is sometimes useful, especially in neuralgias of the fifth. It is usually given in doses of five grains, but, as Ringer and others have shown, the dose may often be increased (up to twenty grains) with advantage. Combinations of nerve-tonics and sedatives are generally necessary, but as the sedative has to be continuously given, its dose must, of course, be moderate. I have found, for instance, in neuralgia of the fifth, the combination of arsenic, quinine, and Indian hemp of great service.

Neuralgic pain is often relieved by local treatment, which is, for the most part, either irritant or sedative; and some remedies combine the two effects. Counter-irritation seems often to exert an inhibitory influence, and to change the action of the nerve-centre; just as a blister around the limb will stop the aura of epilepsy. Either blisters, or sinapisms, or the actual cautery may be employed. Flying blisters may be applied over the tender spots, or by the side of the spine. Chloroform has been injected beneath the skin, fifteen or thirty minims, but, although it may give some relief, it may also cause a troublesome slough. The actual cautery, in mild form, is most effective in spinal neuralgias. The local injection of carbolic acid or of osmic acid has been recommended; they have been employed chiefly in trigeminal neuralgia and in sciatica.* One or two drops of a 1 per cent. solution of osmic acid (in water and glycerine) have been injected at a time. It is said to give immediate relief to the pain; some cases are reported as being cured after about a dozen injections. It does no appear to be entirely free from danger; Jacoby observed palsy of the radial nerve to follow an injection into the arm, as it occasionally follows an injection of ether.

Acupuncture, the introduction of needles into the painful part, is a method employed in China and Japan, the value of which is not great. If, as in the East, the needles are left in for some hours or a day, they cause much pain. Aquapuncture has also been employed; it consists in the injection of pure water into or beneath the skin. Originally it was introduced into the skin, or between the true skin and the epidermis, under considerable pressure, so as to force the water to separate the tissues and make spaces for itself. This method gives much pain and does little good, but the injection of water beneath the skin is innocuous, and occasionally relieves a slight pain.

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* Merces, 'Lancet,' 1885, No. 2; Jacoby, 'Trans. Am. Neurolog. Ass.,' 1885, p. 11; Schapiro, 'Petersburg. med. Wochenschrift,' 1885.

Of external applications that are at once irritant and sedative, chloroform is the most important, pure, or as the Linimentum Chloroformi (B. P.), or a dilution of one part of chloroform with six of Lin. Saponis. Next in value are the ointments of veratria and aconitia. They should be rubbed in until tingling is produced, followed by numbness. The milder tincture of aconite may also be painted on the part two or three times a day, avoiding any sores. Camphor-chloral (camphor and chloral rubbed up together in equal parts, so as to liquefy) may also be applied on lint covered with oil-silk. Both this and chloral will blister if left on too long. Menthol is also a useful application to the skin in the slighter forms of neuralgia. It causes a singular sensation of tingling and coldness, and for the time lessens the pain. It may be employed pure in the solid form, or rubbed up with chloral, or with spirit or glycerine. Preparations of opium (usually oleate of morphia) are of very little value as external applications. A strong ointment of cocaine and lanoline (10 or 15 per cent.) sometimes does good by numbing the skin, but it should not be used at the junction of a mucous membrane and the skin, or when the skin is irritated, or it may excite a troublesome spreading dermatitis. Belladonna is of service if employed sufficiently strong; the extract, diluted with one, two, or three parts of glycerine or vaseline, may be smeared over the skin two or three times a day. The oleate of atropia (5 per cent. solution in oil) may be used in the same way.

Among external applications, that of simple cold or warmth should be mentioned. Each occasionally gives temporary relief if applied continuously to the seat of pain. In most cases heat is the safer and more effectual. Considerable heat is often little felt at the focus of most intense pain. If there is the least suspicion of active neuritis, heat must be applied to the distribution of the nerve with extreme caution, or disastrous consequences may ensue (see vol. i, p. 62). A warm douche through the nose has been strongly recommended by Seeligmuller in cases of supra-orbital pain, supposed to be produced in the frontal sinuses, even when of malarial origin. Ether spray to the spine has also been recommended in various forms of neuralgia. Cold to the painful part is rarely useful, except in the case of neuralgia of the testis, where refrigeration can be more completely effected than elsewhere.

Electricity, properly employed, is an agent of occasional value in the treatment of neuralgia. It may be used in two ways. (1) A strong current, causing, for the time, considerable pain, will sometimes remove the neuralgia at once, no doubt by altering the form of central action, just as does a counter-irritant. The effect is probably only produced in cases of idiopathic neuralgia of slight degree. In some cases, and invariably in active neuritis, a strong application renders the pain worse, and the risk of this effect must be borne in mind. The forms in which it is most likely to be successful are recent cases of hysterical neuralgia, especially when the pain is seated in the joints. To produce this energetic effect either faradism or voltaism may be used. (2) A weak current may be used for its sedative action on the nerves. Either form of electricity may be employed, but the action of the two is essentially different. The voltaic current is the most frequently useful. Authorities are divided on the best method of applying it, but the majority are of opinion (and with this my own experience agrees) that it is best to neglect the direction of the current, and to place the positive pole near the seat of the pain, and the negative in some indifferent situation. If there is reason to believe that the neuralgia is central, the positive pole may be placed, during part of the application, as near as may be to the central termination of the nerve, and, for another part of the time, on the seat of the pain. The strength should be from two to five milliampères, but if the battery is not provided with a galvanometer, the number of cells must be regulated according to its effect on the sensory nerves, so as to cause a slight tingling or burning sensation, not actual pain. The number of cells to be used will vary in different parts, chiefly in consequence of the varying sensibility and resistance of the skin. The patient's sensations are, indeed, often the best guide. For the face, three, and elsewhere five, cells may be employed to begin with, and the current gradually increased until it can just be felt. All sudden variations in strength should be avoided. A well-wetted sponge should be employed as the electrode, and this should be very gently and gradually applied, and very gently removed when the strength is altered. If the faradic current is employed as a sedative, it must be extremely weak (so as to be just felt, but to occasion no pain), and rapidly interrupted. The rapidly recurring slight stimulation of the nerves produces after a time a sedative effect, at first slightly increasing, but after a few minutes distinctly relieving, the pain. Its action is analogous to, and quite as effectual as, the mechanical percussors recently introduced.

The Surgical Treatment of Neuralgia.—Cases of neuralgia are occasionally met with, especially in the region of the fifth nerve, and in persons who have passed middle life, in which every attempt to afford even relief, fails entirely. In such cases the only measure which is likely to be successful in curing the disease is one that interrupts the passage of all impulses along the nerve, from the periphery to the centre. Such impulses, as we have seen, are constantly traversing the nerve; some affect consciousness and are felt as sensations; of others we are usually not conscious, but can perceive them by fixing our attention on the part. But all these are probably only a small part of the impulses which habitually pass up to the cells in which the fibres end, and these impulses seem, in cases of central neuralgia—the common variety in the class now under consideration—to maintain the morbid action of the cells when this has been once set up and fixed in them. If these impulses can be arrested, absolutely and permanently, the disturbed centre is afforded perfect rest from all peripheral influences, and in a large number of cases, with such rest the morbid process subsides. Indeed, it often ceases at once; the shock to the cells, involved in the operation necessary for the arrest of conduction along the nerve, seems at once to inhibit the cells, and in the absence of the previous excitation they do not again resume their normal activity.

The influence of nerve-stretching is seldom permanent enough to produce the desired result; even division of the nerve is uncertain in its effects, apparently because union may take place between the divided extremities which are left in juxtaposition. The only proceeding which has been found effectual and trustworthy is the excision of a short piece of the trunk of the nerve, to the distribution of which the pain is referred. If the pain is referred to more than one division of the nerve, it is usually sufficient to operate upon the branch first and chiefly affected. But a recurrence may take place, and the nerve may afterwards have to be resected above its division. It was at one time supposed that the removal of Meckel's ganglion was essential in some cases,* but this has not been confirmed by recent experience.† The various procedures devised by surgeons to attain the object have been similar in aim, viz. the exposure of the nerve at a point as near the centre as is thought necessary. On account of the tendency for the pain to affect other branches of the nerve, after that primarily affected has been interrupted, the main divisions of the nerve, at one or the other side of the foramina through which they pass from the skull, may have to be divided. Rose‡ even recommended removal of the Gasserian ganglion and has carried out the operation in several cases; while Horsley,§ who asserts that removal of the Gasserian ganglion as a whole is impossible on account of its close connection with the cavernous sinus, advocates division of the nerve between the pons and the ganglion.

Success is a frequent but not a constant result of the operation of neurectomy. In nineteen cases reported by Horsley,‖ the operation had to be repeated in six on account of recurrence of pain; one patient died, in whom he had separated the fifth nerve from the pons; one became demented after repeated operations, which failed to give more than temporary relief. In the others, there was relief up to the time at which the paper was written, this interval varying from a few months to some years. In five cases in which Rose operated on the Gasserian ganglion, complete relief persisted up till the time of the publication of the result, but in three of the cases that time was less than a year. The same is true of a case in which Professor Andrews, of Chicago, carried out the same operation.

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* See a paper by Dr. Chavasse, of Birmingham (Royal Medical and Chirurgical Society, meeting of February 20th, 1884, and discussion thereon).

† Neuralgia will sometimes disappear for a time under strong mental influence. Lefort, for instance, mentions that a most obstinate lingual neuralgia ceased during the siege of Paris, and returned when the anxious time was over.

‡ "Lettsomian Lectures," 'Brit. Med. Journ.,' i, 1892.

§ 'Brit. Med. Journ.,' ii, 1891.

‖ Loc. cit. In these papers will be found described the details of the various operations.

Although operative measures in the treatment of trigeminal neuralgia are thus uncertain in their result, even when carried out on the main trunk and still more upon the peripheral branches, the measure has resulted in complete relief, certainly or probably permanent, in a considerable number of cases in which other measures have failed. With wider experience, a selection of cases may be possible, that will much increase the practical value of the operation.

Nerve-stretching is an operation which produces some of the effects of neurotomy, arresting for a time the conducting function, and effecting what may be called an alterative stimulation of the nerve. If its influence is less lasting, so are also its inconvenient effects, and, as it is sometimes successful, it may often wisely precede division of the nerve. It has been recommended that strong traction should be made on the central end, but it is doubtful whether this has much influence, and, in the case of the fifth nerve, it is somewhat perilous.* Recent literature abounds with records of cases of apparent cure from nerve-stretching, even when the neuralgia seemed to be central. For instance, a severe intercostal neuralgia of twenty years' duration is said to have been cured by stretching the terminal branches of two or three of the nerves.† Intensely severe neuralgia of the third division of the fifth—the pain being in the ear, temple, lower jaw, and tongue,—of five years' duration, is said to have been cured by a single stretching of the lingual nerve in the tongue.‡ In this and many other cases the pain did not immediately cease, but gradually subsided in the course of one or two weeks. In some rare cases, in which the sheath of an accessible nerve can be felt to be greatly thickened, the nerve has been exposed, the sheath opened carefully, and the nerve for some distance separated from it. This operation has been successful in arresting the pain, especially in traumatic cases.§

Ligature of arteries has been confined to that of the carotid, as a last resort in cases of neuralgia of the fifth nerve. All that can be said to justify so serious an operation is that it has sometimes, but very rarely, been successful. Compression of the carotid occasionally cuts short an attack of pain, and the repetition of this treatment, continued for a long time, has even produced permanent alleviation.

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* There is evidence that too vigorous stretching of the second division of the fifth nerve has caused destructive inflammation of the eyeball, probably by the mechanism of inflammation of the Gasserian ganglion (Nicaise and Tillaux, Soc. Clin. de Paris, March 9th, 1882).

† Nussbaum, 'Aerzt. Int.-bl.,' 1878, No. 53.

‡ Le Dentu, 'L'Un. méd.,' 1881, vol. ii, p. 766.

§ An instance is recorded by Le Fort, 'Soc. de Chir.,' July 26th, 1882, in which the median was surrounded by a dense sheath of connective tissue, due to inflammation produced by a gunshot wound.

An attempt to estimate the true position of the surgical treatment of neuralgia is unfortunately beset with almost insurmountable difficulties, due to the fact that after many operations the pain has ceased, or has been much lessened, for a time, but has afterwards returned, and many cases have been published before sufficient time has elapsed to permit an opinion to be formed of the permanence of the effect. Moreover, the numerous cases in which there has not been even temporary relief are seldom published. In some instances, operation after operation has been submitted to by the patient under the urgent compulsion of continued suffering, and the records of many cases illustrate very strikingly the need for caution in drawing any inference from transient relief.*

VISCERAL NEURALGIAS.

The internal viscera of the thorax and abdomen are sometimes the seat of neuralgic pain. Such neuralgias are described in full in the works that deal with the diseases of these organs, and this arrangement is convenient, since the principles of diagnosis involve a differential discussion of the symptoms of organic disease of these organs which would be out of place in the present work. A brief outline of the general facts may, however, be given here.

Most of the organs in which these neuralgias are felt, receive their chief nervous supply from the sympathetic system. Little sensation attends their normal function. Although it cannot be doubted that afferent impressions are constantly passing to the cerebro-spinal centres, these fail to affect consciousness under normal circumstances. But repeated attention may vastly increase the sensitiveness of the perceptive centres to such impressions, and from such increase arises a large amount of the discomfort of those patients who are termed "hypochondriacs"—correctly, in so far as the organs which lie below the rib-cartilages disturb their conscious life. But the sensation from the viscera may also amount to actual pain, in consequence of the afferent impressions being abnormal, owing to organic disease and varied functional disturbance. Pain may also be felt apart from either of these causes, and such pain is called "visceral neuralgia." Often we cannot tell to what extent it is the result of a local abnormal condition of the nerves of the organs, and how far it is central. It is probably local in causation to a larger extent than in the case of the cerebro-spinal forms of neuralgia, but the same general pathological laws doubtless underlie the two. The difficulty of investigation depends not only on the fact that the organs are concealed from direct examination, but also on the circumstance that they receive their innervation from two sources, from the sympathetic, and from the cerebro-spinal centre by the pneumogastric and spinal branches; and we do not know what share these sets of nerves respectively take in the production of visceral pain. There is, moreover, reason to believe that not only the nerves in the viscera but also the nervous plexuses outside them may be the seat of neuralgia. The difficulties of investigation are greatly increased by the fact that pain may be the only symptom of organic and of functional disease, and it is certain that many examples of the former have been included among the visceral neuralgias by some writers on the subject.

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* The following cases are illustrations of this. In one instance, a man, at the age of forty-five, had his first attack of neuralgia in the third division of the fifth nerve. Four teeth were extracted from the lower jaw and then one from the upper, and, as the pain ceased for three weeks, the case was published as cured. The pain returning, resection of the alveolar process was performed; the pain ceased for five months, and the case was again published as cured. After a relapse, the inferior alveolar nerve was excised, and freedom for some time was followed by a return, for which the carotid artery was tied, but the effect of even this was not permanent (J. C. Hutchinson, 'Am. Med. News,' 1885, p. 395). The same author relates a case of neuralgia of the fifth, in which the following operations were performed without success:—an incision through the skin above the ear, division of the supra-orbital and infra-orbital nerves, excision of half an inch of the supra-orbital, ligature of the carotid, destruction of the nerve in the infra-orbital canal. In a case, recorded by Schupper, of neuralgia of the fifth with reflex spasm in the face, the first procedure was scarification of the outer and inner surfaces of the upper jaw; this failing, the surgeon excised the infra-orbital nerve; this likewise failing, he excised the superior maxillary nerve in the spheno-maxillary fossa; there being no relief, he removed the peripheral segment of the same nerve from the base of the orbit. The pain then migrated to the third division of the fifth: the inferior maxillary nerve was therefore excised. An interval of freedom was followed by a return of the pain, and the common carotid was tied. The pain continuing, the facial nerve was divided, and on account of severe hæmorrhage, the external carotid and temporal arteries were tied, and the patient is said at last to have been "cured."

Most forms of visceral neuralgia are more frequent in females than in males, and may be disposed to by the same inherited tendency that produces the cerebro-spinal forms. The general causes of the two are also, for the most part, similar. In women, anæmia and hysteria are especially prominent. They may also result from causes that have a local action, and this fact adds not a little to the obscurity of their diagnosis. The essential symptom is pain, which varies greatly in character, sometimes dull and diffuse, sometimes sharp, circumscribed, lancinating or burning. It usually presents paroxysmal exacerbations, and is sometimes actually intermitting. I have known paroxysms of gastric pain to alternate with headache. The exacerbations may be apparently spontaneous, or may be produced by various stimuli, especially by those concerned in the functional activity of the organ. We ought not, however, to include among the neuralgias (as some have done) cases in which pain is confined to periods of functional activity. It is doubtful whether, in such cases, the affection is ever a pure neuralgia.

The diagnosis of visceral neuralgias is difficult, not only in the recognition of the actual cause of the pain, but also in that of the organ or structure from which it proceeds. The first and chief element, however, is the exclusion of organic disease, by every method of investigation that can be made available. If functional disturbance exists, the affection can only be regarded as neuralgic when the pain is not related to the disturbance of function either in time or in degree.

The treatment of visceral neuralgias must be based on the same general principles as that of the cerebro-spinal forms. It consists in the removal of causes, general tonic treatment, abundant rest, and the use of sedatives. The mode of employment of the latter differs in each case, and in each, also, special measures are necessary, related to the function of the organ concerned.

SPECIAL FORMS.—Of the intra-thoracic organs, the lungs do not appear to be the seat of neuralgia, although the pain of "pleurodynia," already described, is probably due to an affection of the pleural nerves. The only important cardiac affection of this class is the disease known as "angina pectoris." Severe nerve-pain is a prominent symptom of the disorder, but its peculiar and special characters and associations prevent its inclusion among the forms of pure neuralgia.

The most important visceral neuralgias are those of the abdominal organs. They are usually local and well defined, but occasionally abdominal neuralgic pain varies in its seat, and is felt now on one side, now on another. That of the stomach (*gastralgia, gastrodynia*) is one of the best-marked forms. It is frequent in anæmia and hysteria, and a special form constitutes the gastric crises of ataxy. The pain is felt at the epigastric region, and, like most gastric pains, passes through to the back. Pressure does not usually increase it; often, indeed, it relieves the more intense suffering. There is generally a constant dull pain, with more acute exacerbations. It may be most intense when the stomach is empty, and may be relieved by food; it is then possibly due to the morbid action of the nerves or centres from which, in health, the sensation of hunger arises. Or the pain may be increased by food, and in such cases food is often vomited as soon as it is taken. Appetite may be absent, lessened, increased, or perverted. The relation of the condition of appetite to the effect of food in relieving or increasing the pain deserves further study.

The stomach is readily influenced by sedatives, and their administration by the mouth forms an important part of the treatment. Other agents are sometimes useful. In the form that is relieved by food, oxide of silver is often of great service. When there is anæmia, the combination of subcarbonate of bismuth and saccharated carbonate of iron, given before food, is frequently effective. No sedative, however, is on the whole so useful as cocaine, of which half a grain may be given when the pain comes on. By repeatedly relieving the pain in this manner, the disease is often cured, apparently by the rest thus given to the nerves of the mucous membrane.

The frequency of intestinal neuralgia (*enteralgia*) has probably been exaggerated. We are not justified in regarding as enteralgia either vague abdominal pains, which are not increased by peristaltic action, or pain that occurs only when the intestines are in energetic action, or in which there is conspicuous disturbance of the mucous membrane. If these are excluded, the cases of enteralgia become extremely rare, and the history of the affection has yet to be worked out. There is, however, one part of the intestine which is undoubtedly the seat of neuralgic pain,—the lower part of the rectum. This form occurs in both sexes as a deeply seated pain above the anus and coccyx, more or less constant, but with severe exacerbations, apparently due to spasm. It is usually effectually relieved by suppositories. In some cases, indeed, it is probably a primary spasm, and I have known it to be produced in a child by the use of senna as an aperient.

Of neuralgia of the liver, *hepatalgia*, much the same may be said as of enteralgia. Deep-seated pain is occasionally felt in the position of the organ, sometimes diffuse and dull, sometimes sharp and lancinating, but the cases in which we can be sure that it is truly neuralgic are extremely few. In some cases, such pain may be due to disturbed function of the organ. Disturbance of function has been regarded by some writers as a consequence of neuralgia. It cannot be denied that severe nerve-pain may cause such reflex derangement of function, just as it causes vaso-motor disturbance in other situations. But the frequency with which pain is the first evidence of disease outside the nervous system, which afterwards causes other symptoms, renders it necessary to exercise great caution in regarding such cases as primarily neuralgic.

The existence of neuralgia of the spleen is not well established, but occasionally, especially in cases of hysteria, there is deeply seated pain and tenderness in the position of the organ, apparently situated in it or in the nerve-plexuses in its vicinity.

The kidney, and apparently the ureter, may be, in rare cases, the seat of the neuralgic crises of tabes, analogous to the more frequent gastric crises; but the occurrence of primary *nephralgia* is a still more rare event, and can hardly be regarded as proved. Many cases of supposed renal neuralgia have certainly been due to the passage of a calculus. I have, however, met with one case in which paroxysms of pain in the renal region had occurred at times during forty years without any indication of a calculus, and it seemed on the whole probable that the pain was of nervous origin.

Neuralgic pain which cannot be referred to any organ is sometimes felt within the abdomen. Such pain is diffuse, varies in intensity, is not increased by pressure, and is not related in time to the functional activity of the organs, or in degree to their disturbance. It is generally central in position, but may be felt above or below the umbilicus, apparently seated in the sympathetic nerves, although without such a relation to their plexuses as to permit definite localisation.

The female generative organs are frequent seats of pain and tenderness. Tenderness in the region of the ovaries is extremely common in hysteria and conditions of nervous weakness; sometimes there is much spontaneous pain in this situation, deeply seated, and aching or burning in character. This pain may exist when no evidence of organic change in the ovary can be detected, and appears then to be a pure neuralgia; but in most cases the tenderness is extensive, and exists in the vicinity of the ovary as well as in the organ itself, so that it is probably due, at least in part, to the abundant nerve-plexuses with which the ovary is surrounded. The uterus is also the seat of spontaneous pain and of tenderness, apart from organic disease or of displacement. These symptoms may occur in the young and also in later life, even when the involution of the organ is complete. They are often associated with neuralgic pains elsewhere—in the ovaries, and especially in the spine, including the sacrum. By some writers the pains of menstruation, when no organic cause for them can be discovered, are regarded as neuralgic.

Of the male organs of generation, that which is most frequently the seat of neuralgic pain is the testicle, but even here such pain is comparatively rare. It is important to remember, however, that inflammations of the urinary passages are not uncommon causes of neuralgia seated in the adjacent spinal nerves.

MIGRAINE: PAROXYSMAL HEADACHE.

Migraine is an affection characterised by paroxysmal nervous disturbance, of which headache is the most constant element. The pain is seldom absent and may exist alone, but it is commonly accompanied by nausea and vomiting, and it is often preceded by some sensory disturbance, especially by some disorder of the sense of sight. The symptoms are frequently one-sided, and from this character of the headache the name is derived, the Latin "hemicrania" (still often employed) furnishing the French migraine, the German migrān, and the English megrim.[*] The French word is that most frequently employed, and is, on the whole, the most convenient. On account of the associations of the pain, it has received the popular names of "blind headache," "sick headache," and "bilious headache," the latter being derived partly from the fact that bile is often vomited, partly from the old humeral pathology which regarded the bile as one of the chief morbific fluids of the body. The disease is often associated with high intellectual ability, and many distinguished scientific men have suffered from it, and have supplied more careful observations of the subjective symptoms than we possess of any other malady. Amongst the sufferers may be mentioned the celebrated Dr. Fothergill, Marmontel, Haller, Wollaston, Du Bois Reymond, Sir Charles Wheatstone, Sir John Herschell, Sir George Airy, and his son, Dr. Hubert Airy.*

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[*] The English word has been developed by the following steps, going backwards: megrim, megrene, emigranea; Low Latin, hemigranea; Latin, hemicranea, hemicranium; Greek, ἡμικράνιον (Skeat).

Etiology.—Females suffer from migraine more frequently than males, but their preponderance is not great, and has been much exaggerated by some writers. The affection usually commences in the first half of life. One third of the cases begin in later childhood between five and ten; about two fifths between ten and twenty, and most of the others between twenty and thirty. The maximum periods are late childhood, puberty, and early adult life. Now and then the disease commences after thirty; I have met with one well-marked case which began at sixty. In the ratio between the sexes, and the large number of cases which commence at the time of puberty, the disease resembles another paroxysmal neurosis, epilepsy, and we shall see that there are other points of resemblance between the two diseases. Migraine is strongly hereditary; in more than half the cases inheritance can be traced, and it is usually direct, *i. e.* other members of the family (very often a parent) suffer from paroxysmal headache. Now and then the inheritance is indirect; relatives suffer, not from migraine, but from some other neuroses, especially pure neuralgia and epilepsy. For instance, a woman aged forty-six had suffered for many years from migraine (paroxysmal headaches with dimness of sight and vomiting). One of her children was epileptic, and her mother had been epileptic and insane. I once had under treatment a brother for migraine, and his sister for epilepsy. One patient's brother suffered from paroxysmal headache, her father from severe neuralgia, and her father's brother was insane. Occasionally, migraine seems due to the inheritance of the gouty diathesis: a father may suffer from gout, and his son from migraine. A similar transformation may occur in the course of the disease; migraine, commencing in early life, may cease when distinct gout is developed. Trousseau has emphasised (but also exaggerated) this relation by saying that "migraine and gout are sisters."

When migraine begins early, no immediate cause can usually be traced; but when the disease begins later, its occurrence, or in cases that begin earlier its exacerbation, is related to influences that depress and weaken the nervous system, either directly or through the general health. Such causes are excessive brain-work (especially combined with anxiety and loss of rest), over-fatigue of all kinds, work in hot and crowded rooms, anæmia, over-lactation, and the like. I have seen one case that appeared to date from attacks of ague, which the patient had had in early life.

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* Dr. H. Airy has given a very interesting account of his ocular symptoms in the 'Philosophical Transactions' for 1870. The best systematic account of the disea is that of Dr. Edward Liveing ('Megrim, Sick Headache, and some allied Disorder London, 1873).

Symptoms.—The essential feature of migraine is paroxysmal headache, but a large number of the patients present also other sensory symptoms in association with the headache, and, in rare cases, these sensory symptoms occur alone, without headache. These associated symptoms are so peculiar and striking, that undue importance has been given to them as the characteristics of the disease. They are often inconstant; even the same patient may have some headaches with, and others without, these accompaniments, or may have simple headaches at one period of his life and the more complex series of symptoms at another period. The simple headaches have the same characters, and occur under the same causal conditions of heredity, &c., as those in which there are in addition other sensory symptoms.

The characteristic feature of the symptoms is their paroxysmal character. During the intervals most patients are free from any symptoms of nerve derangement, although a few suffer from slighter headaches of a different character, occasional or continuous. The precise elements that make up the paroxysmal seizure vary in different cases, and often even in the same individual. Headache, as already stated, is the most constant, and is very seldom absent; next in frequency are nausea and vomiting, then some disturbance of vision, affection of speech, disturbed sensation in the limbs, and vaso-motor derangement, while the least common are motor symptoms in the limbs. The frequency of vaso-motor derangement will be variously estimated, however, according to the phenomena included in the term, and if a simple change in frequency or tension of the pulse, or in the colour of the face, is included, this disturbance becomes one of the most frequent features of the attacks.

But the above order of frequency is not that in which the symptoms occur during an attack. The various sensory accompaniments of the headache usually occur first, then comes the pain, and after the pain has lasted for a time, nausea occurs, followed by vomiting, and this often ends the attack. Vaso-motor disturbance may be present in some form throughout an attack, or may come on only towards the close. When sensory disturbance is absent, the pain is the first symptom.

Various influences will induce a paroxysm. Fatigue and excitement are the most common. Digestive disturbance is a potent cause, and sometimes a particular article of diet will always induce an attack, but most of the sufferers who are thus susceptible learn by experience the dietetic errors that are efficient, and carefully avoid them; hence, as an actual fact, it is not very common for attacks to be thus induced. Frequently, after the usual interval between the attacks has nearly elapsed, a slight error in diet is sufficient, although, soon after the patient has had an attack, even actual indigestion has no effect. The influence of stomach derangement is also exaggerated on account of a misconception of the significance of the vomiting that so often occurs. The bile that comes up is thought to be a proof of "biliousness," when its rejection is merely the result of the repeated vomiting. In some patients exposure to cold will bring on a paroxysm. Another occasional excitant is a visual impression, such as watching moving objects or seeing some peculiar kind of motion. Over-use of the eyes may also bring on an attack. In some patients a bright light, or a sudden change of light, has the same effect, and so has a loud noise or a peculiar odour. Indeed, it seems as if a peculiar habit may become established, so that a certain sensory impression will always induce a paroxysm.

Premonitory symptoms are present in some cases, but are less frequent when there are accessory symptoms than in the attacks that consist of pain only. The day before an attack the patient may complain of heaviness in the head, or of slight pain, or of somnolence. When attacks consist of simple headache, the patient often wakes up with it. When sensory symptoms occur first, these often begin quite suddenly. The patient, for instance, may feel perfectly well, when he is suddenly conscious of some disturbance of vision, of a bright spot, for instance, on one side of the field of vision, which slowly enlarges and spreads, becoming darker in the centre as it extends, and changing its round outline into an angular form. Or he may suddenly be conscious of dimness of sight towards one side, which increases in extent and intensity until one half of each field is blind. Or the first symptom may be tingling in one hand, which spreads up the arm. Such sensory disturbance lasts for ten, twenty, or thirty minutes, and then passes away, and as it subsides, headache comes on; it is usually of great intensity, commences at one spot, and slowly spreads. After a few hours the patient feels sick, and the nausea slowly increases and at last ends in vomiting; then the sufferer goes to sleep for an hour or two or for the night, and wakes up well. These various symptoms are generally unilateral in distribution. The pain is sometimes bilateral, but it is then greater on one side than on the other. The symptoms may now be considered in detail.

Visual disturbance occurs in at least half the cases, and is the earliest symptom of an attack. It may consist in partial loss of sight, or spectral appearances, or both. The unilateral character of visual symptoms is always manifested as affection of the corresponding halves of both fields of vision.

Loss of sight is always imperfect. There may be sudden general dimness of vision, or there may be a lateral limitation of the field, extending from one side and not reaching the centre, or commencing first on one and afterwards on the other side, during the same attack. The resulting hemianopia may be complete. In other cases, the first change in vision is a spot of dimness of sight, lateral or central in position, which gradually increases in size and extends towards the periphery; when lateral in position it usually does not pass beyond the middle line, so that from this also hemianopia results. Very rarely the spot is situated in the upper or lower parts of the field, and may cause a form of transverse hemianopia. The degree of loss varies; it is often described as a "cloud," but the darkness may be noticeable only when a bright light is looked at. As the dark spot increases in size it often clears in the centre. When spectral appearances occur, they may commence as a bright spot, gradually expanding, or they may develop out of this area of dimness. In the latter case, as the dark spot increases it becomes luminous at the periphery and expands, so as at first to form a circle; but, if lateral, it may break at the middle line into a crescent. In some cases it spreads over almost the whole field of vision. When a luminous spot is the first change and this expands, it may become dim in the centre. Very commonly the outer edge assumes a zigzag shape with prominent and re-entrant angles, like the ground plan of a fortification, and hence called "fortification spectrum." At one part it becomes fainter and ceases, so that there is a break in the outline. The outer boundary is the most brilliant, and is often limited by colour; inside, the luminosity extends for a little distance, gradually becoming fainter. Very frequently within the bright outline, however it arises, there seem to be luminous particles in rapid irregular movement. The spectrum increases with the blind area, gradually becomes indistinct, and disappears at the periphery of the field. These visual phenomena always affect the field of vision of both eyes, although the patient often imagines that the phenomena observed on one side are seen with one eye only. They may present considerable variation in the same case. Thus one patient described sometimes hemianopia, sometimes coloured lights, sometimes merely a sensation as of moving water before the eyes. Many patients experience only slight and vague ocular symptoms, such as sparks or mere flashes of light. Very rarely there is double vision.

Disturbance in the function of the other special senses is exceedingly rare, but a few cases are on record in which phenomena have been observed in hearing and taste similar to those in vision. Thus there has been one-sided deafness followed by a noise in the ear, or loss of taste followed by a subjective sensation. Transient tinnitus is occasionally observed in the subjects of migraine, without connection with the attacks.

Other sensory symptoms are felt in the limbs, face, throat, tongue, and adjacent parts, but these are far less frequent than is the visual disturbance. In the limbs, the sensation is felt chiefly in the arm, very seldom in the leg. It may occur alone as the first stage of the attack, but is more often associated with the visual phenomena, succeeding the latter but commencing before the affection of sight has quite ceased. In character, the disturbance of cutaneous sensibility is very similar to that of vision, allowance being made for the difference in the character of the function. There is the same combination of sensory irritation and sensory loss. Tingling in the skin, or "pins and needles," is felt in some part of the hand, as the fingers, or in the wrist, and as it spreads it is succeeded by numbness and defect of sensibility, sometimes amounting to actual anæsthesia. In other cases the numbness occurs first, and is succeeded by tingling. The sensory disturbance in its double form may pass up the limb from the extremity, and the leg may be affected after the arm, just as in the sensory aura of epilepsy. Sometimes, instead of a gradual extension, the tingling passes from one part to another at a distance. It is generally confined to one side, but sometimes is felt first in one arm and then in the other. A sensation in the lips and tongue is generally secondary in time to that in the limbs; it rarely exists alone. It may be felt in the cheek, lips, tongue, or fauces, on one side or on both; the side is usually the same as that of the limbs, but the sensation may be bilateral in the lips and throat, and unilateral in the limbs. The duration of these sensations is generally about ten or fifteen minutes. Occasionally slight motor weakness accompanies the tingling, just as it may do in the sensory discharge of epilepsy. For instance, the attack in one patient commenced by dimness of sight, and this was followed by tingling in the fingers of the left hand; the sensation passed up the arm to the shoulder, and was followed by weakness of the limb for about ten minutes; then headache came on in the right occipital region, and lasted for twelve hours.

Motor symptoms in the limbs, with or after the sensory disturbance, are usually confined to such transient weakness as occurred in the case just mentioned. If any motor spasm is present, the case usually diverges very much from the type, and sometimes is of such a character as to render it doubtful whether it should be classed with migraine or not. In one patient each attack of headache was preceded by sudden tingling in the calf, followed by painful cramp in the calf muscles, lasting a few minutes only. The same patient, however, had at other times attacks in which her face suddenly became crimson, sharp pains occurred in the head, and seemed to pass down the side to the leg, which was then "drawn up" in spasm for a few minutes.

Difficulty in speech, transient aphasia, is another occasional symptom of the commencing attack. If there are sensory symptoms, these are almost always right-sided, and are situated, in most cases, in the right arm. If there is visual disturbance, this also is in the right half of the field, but I have only once met with aphasia in association with an affection of sight alone. The rule of the right-sided association probably does not hold good of left-handed persons, and it is not quite absolute in other cases. I have met with one case, in a right-handed man, in whom the attack began with left-sided hemianopia, followed by tingling in the left foot, which passed up the leg and side to the mouth and tongue, and then the speech was deranged, the words of a sentence "coming out in wrong order." The common character of the defect is that there is a difficulty in finding the right word, or a use of wrong words in both speaking and writing, or very rarely a total inability to speak. The latter suggests motor aphasia (see p. 114), but the more common form has the character of the sensory variety. In one case the affection of speech was clearly of this character; the patient was, for a few minutes, completely "word-deaf;" when spoken to, she heard the sound perfectly, but could not tell what was said. A few similar cases are on record.*

Slight mental change occurs in some patients during the attack. Emotional depression, restlessness, or confusion of ideas are the most common; sometimes there is transient loss of memory. Brief stupor, without complete unconsciousness, sometimes occurs soon after the onset of an attack, without interrupting the progress of the sensory disturbance. Among other peculiar conditions that have been described is a sensation of "double consciousness" or a vivid recollection of events long past. The mental change may be the earliest symptom, or it may succeed the affection of sight, or occur when the sensation in the limbs ascends to the head.

Giddiness is not a frequent symptom of the attacks themselves. It is usually a vague sense of defective equilibrium, rarely amounting to definite vertigo. It varies in time, being occasionally early, but more often succeeding the sensory disturbance, and it often accompanies the headache. Any one of this series of symptoms may precede the headache, or all may be absent. Even when many of them occur, their duration is short, varying from fifteen to thirty minutes, before they give place to the pain in the head.

The headache is not only the most constant symptom, it is also the most distressing. It is not, however, so alarming to the patient as the sensory symptoms are, especially in those who are unaccustomed to them, and in whom they occur at long intervals, or, for the first time, in adult life. The pain has generally recurred during many years, and the sufferer knows what to expect and how best to endure it. It varies much in degree, but is very seldom trifling. Often it has a characteristic course, uniform in the same patient; it begins gradually, slowly increases to a considerable degree of intensity, and after a variable time it subsides, sometimes slowly, sometimes rapidly. It is usually an acute pain at the onset, and may remain so throughout, or may assume a duller character as it spreads. Movement, noise, and light usually increase its intensity. Sudden stooping also makes it worse, but the patient is, nevertheless, most comfortable in the recumbent posture, and suffers more when he attempts to sit up or to stand. In most cases the headache begins on one side, and in many it is confined to one side; in others it becomes general, so that the name, while founded on a common characteristic, has no universal significance. When the pain begins at one spot it most frequently is in the temple, and is confined at first to a small area that can be covered by the tip of the finger. In other cases it begins in a small spot on the forehead, or in the forehead and eyeball, seldom in any limited area at other parts of the head. When thus limited at first, it seems generally superficial, but often has a boring character, as if some instrument were being forced into the skull. After a time the pain may spread through a considerable part of one side of the head, and not unfrequently through both sides. Sometimes it begins at the back of the head, in the occipital region on one side, and may then extend forward to the temple. It occasionally commences in the middle of the head, and spreads down one side. From the back or side of the head, the pain may pass down the side of the neck and even into the arm. Occasionally it spreads from one side to the other, and may then cease on the side first affected. In one instance in which it had this course, it ultimately ceased on the second side, and recommenced, in slight degree, on the side first affected before finally passing away. Even when the pain is limited in area and superficial in character, there is seldom any local tenderness, but now and then extensive pain over the head may be accompanied by some general tenderness of the hairy scalp, which continues for a time after the headache has ceased. When the pain is unilateral and felt over a considerable area, it is generally on the side opposite to the peripheral symptoms. It usually comes on as the sensory disturbance is declining, and always lasts for several hours, often for the rest of the day. It is not always constant either in character or seat, but when inconstant in seat the patient has certain kinds of headache which maintain their characters, although sometimes one, sometimes another comes on. In other cases, at a certain period of life, there is a change in the seat and character of the pain. Thus in one case the pain for many years was occipital, but afterwards it was always limited to one frontal region.

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* It is probable, and in harmony with the other symptoms, that the primary disturbance of speech is "sensory aphasia," and that when there is "motor aphasia," this is analogous to the weakness of the arm that accompanies the sensory discharge.

Nausea accompanies the headache in a large number of cases, but often does not commence until the pain has reached its height. It is attended with a total inability to take food, and food that is taken is not digested, apparently from a derangement of the gastric secretions. The nausea often results in vomiting, but retching is still more frequent. Neither occurs, as a rule, until after the pain in the head has reached its climax, and often not until the pain is subsiding, and then it may terminate the seizure, even when nothing is ejected. It is common for the headache to commence in the morning, for nausea to come on in the course of the day, and vomiting in the evening. Sometimes the vomiting begins earlier; in one case, in which the visual disturbance was unusually prolonged, lasting two hours, the vomiting commenced before it had ceased and before the headache. If it occurs while the headache is severe, the vomiting does not influence the pain. When there is no headache there is no vomiting. The condition is acompanied with great prostration, and the patient is very much like one suffering from sea-sickness.

The most common vaso-motor symptom is pallor of the face at the onset of an attack, and often throughout its course. The extremities also are usually cold. The face is not only pale, but has a "pinched" or "drawn" expression. The accessible arteries may be sometimes felt to be contracted. In some cases this aspect continues throughout the attack; in others the pallor gives place to flushing as the pain in the head develops, and there may even be general perspiration. Rarely the face is flushed from the first. Still more rarely there is a conspicuous difference in the aspect of the two sides of the face; there may be pallor only on one side, and in addition the eye may be retracted, the conjunctiva injected, and the pupil small; as the paroxysm goes off this condition may be exchanged for one of hyperæmia, the face becoming warm, the ear red, and the pupil resuming its normal size (Du Bois Reymond, Morselli). The latter must be referred to diminished action and the former to increased action of the sympathetic fibres, although in over-action the retraction of the eye and redness of the conjunctiva are exceptional. Unilateral sweating has also been observed. At the end of an attack, in which the final dilatation of vessels has been marked, puffiness of the scalp has been observed in rare cases, and even ecchymoses at the seat of the most intense pain. It must, however, be remembered that such unilateral sympathetic symptoms are not only exceptional, but extremely rare. In a woman, aged fifty, liable to right-sided migraine from youth, the right temporal artery was harder and more rigid than the left, and the right cornea presented an arcus senilis twice as broad as that on the other side (De Giovanni). Œdema of the optic disc has been said to occur during the paroxysm (Möllendorff), but as a rule both during the attack and in the intervals the appearance of the discs is perfectly normal. Occasionally, retardation of the pulse occurs during the paroxysms. In one of my patients the pulse always fell to about 56, and a retardation to 40 has been observed.

The termination of the paroxysm is sometimes attended not only by vomiting, but also by some secretion, copious diuresis or perspiration. The pain passes away gradually, very seldom quickly. The most frequent termination is by sleep. During the height of the attack, the patient may be drowsy and doze, but this brings no relief; as the pain is subsiding, however, he goes to sleep, sometimes for a short time only, and wakes up free from pain. The duration of the headache is always several hours; often it commences in the early morning and lasts the entire day; in severe cases it may last for several days.

Varieties.—The cases in which the collateral disturbance is absent, and the attacks consist only of pain and sickness, are very common. The pain has the same characters as in the cases with other sensory symptoms, and may be attended by the same vaso-motor disturbance. In one patient, for instance, the pain began in one eye and the supra-orbital region, and commenced alternately on each side; from the place of commencement it extended over the whole head, and into the throat. It is very common for two kinds of headache to occur, and for one only to be accompanied by other sensory symptoms. Sometimes one is attended with vomiting, and the other is not. One patient, for instance, sometimes had attacks of pain in the forehead and temples, and at other times pain at the top and back of the head, and only the latter were attended by sickness.

Migraine in adults is not commonly attended by any alteration in the temperature of the body, but in children there may be considerable pyrexia, which impresses a special and sometimes misleading character on the attack. Thus, a child at the age of two became liable to attacks which lasted only a few hours, and recurred at intervals of two or three months. In each there was severe one-sided pain in the head; the temperature rose to 102° or 103°; sickness came on, the child went to sleep, and woke up well.

The sensory symptoms of migraine, as we have seen, sometimes occur without headache, or, more frequently, with headache that is so slight as to cause the patient no distress, and to lead him to place no weight upon it in his description of the symptoms. These cases are of great importance, because their nature is often misunderstood. In one case, with characteristic visual disturbance (an expanding luminous spot and hemianopia), the only discomfort was that a cough or deep inspiration caused momentary pain over the eyebrows during two or three days after an attack. A more common history is that some attacks are complete, consisting of sensory disturbance and headache, while in others the former occurs alone. In rare cases the sensory disturbance or aphasia generally occurs by itself, headache being seldom or never associated. Lastly, some sufferers from migraine often have slighter and variable sensory disturbance, evidently of the same nature, although not of the same form, as that which precedes the headaches. One patient, for instance, with characteristic headaches preceded by hemianopia, complained of bright stars before the eyes whenever she had looked at a brilliant light, and sometimes one of these stars, brighter than the rest, would start from the right lower corner of the field of vision, and pass across the field, generally quickly, in a second, sometimes more slowly, and when it reached the left side would break up and leave a blue light in which luminous points were moving. These sensations were not succeeded by headache, although the pain always followed the hemianopia. Aphasia does not often occur without headache; such attacks may be very puzzling unless their nature is suspected. I have once known ocular spectra to develop after the headache had commenced, and also tingling in the limbs of one side, from the hand to the elbow, and foot to the knee, to follow characteristic attacks of pain. Very rarely vomiting may precede the headache.

I have met with one curious case in which visual disturbance, exactly such as precedes attacks of migraine, occurred frequently during many years as an isolated symptom; at no time was there any pain. The patient was a man of sixty, the subject of chronic bronchitis and some loss of memory, but with no other indications of nerve disease. The visual spectrum was generally a brightly coloured zigzag; sometimes it had the shape of a broken oval, but more often a long comet-like form, commencing on one side of the field of vision and extending downwards. Rarely there was a luminous disc, which would ascend, break into a four-leaved object, and then disappear. A noteworthy feature of the illusion was that the angular spectrum was sometimes related to the image of some seen object. Thus, on one occasion, a plate, which was before the patient as he sat at table, appeared surrounded by the coloured angular spectrum.* A similar relation has been described to me in other cases; a zigzag spectrum would fix itself for a short time around some object at which the person was looking. A more general phenomenon of the same class is the greater brightness of the subjective sensation when the eyes are open than when they are closed—the morbid activity is intensified by the impulses from the optic nerves.

Sensory disturbance in the limbs does not often occur without headache, but occasionally the headache is slight. Sometimes an attack is apparently rendered abortive in consequence of some drug that is taken, such as bromide,—as in the following case, which deserves description on account of the illustration it affords of the deliberate march of the sensory disturbance. A gentleman, the subject of migraine, was working with the microscope one afternoon, when his sight became dim so that he could only just read large print, and continued so in spite of a drachm of bromide. After two hours, tingling suddenly commenced in the left thumb, and spread to the fingers, and then was felt in the middle of both lips, in the tip of the nose, and beneath the tip of the tongue. Then it was felt in the left arm near the axilla, and in the left side of the fauces and of the face over the lower jaw. A few minutes later it involved the fauces on both sides and the palate, and caused an unpleasant sense of constriction. It then ceased and headache came on. Another attack began in the same manner, but after being felt in the fingers and lips and tongue, it became intense at the wrist, and ceased in the lips; the sensation passed up the ulnar side of the forearm and then ceased, but afterwards recurred in the cheek and side of the throat.

The variations in the character and seat of the pain which have been already mentioned are sometimes such as to make the case deviate definitely from the type. This is especially marked in cases in which the pain presents, in its extreme development, an approximation to neuralgia. It may, for instance, spread from the head down the cervical spine, or the sides of the neck to the shoulders. In one patient, in whom the pain (preceded by the usual visual symptoms) was chiefly over the posterior half of the head on the left side, one severe attack was followed by an eruption of herpes over the left half of the occipital bone, which had definite neuralgia for its sequel. Both before and after this, the commencing subsidence of the pain in each attack was commonly attended by the occurrence of small slightly raised "lumps" in the skin of the same region, about the size of a pea. I have met with tenderness of the scalp after the pain, in other cases, in which it began in one temporal region, after hemianopia.

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* The patient was a mechanical draughtsman, and he had a small book full of drawings of the appearances that he saw.

Course.—The interval between the attacks varies in different cases; it is usually between a fortnight and two months. It is generally about three or four weeks, but the periodicity is not often exact. In a case mentioned by Trousseau, however, the attacks occurred each fortnight almost to an hour. In women they often occur about the menstrual period, generally after the catamenia have ceased. The intervals are, on an average, shorter in the cases in which there is only pain than in those in which there are associated sensory symptoms; and when these symptoms occur alone, without headache, the intervals generally amount to several months. The intervals are doubtless rendered more irregular than they otherwise would be by the influence of exciting causes, effective when the usual period has nearly elapsed. In the intervals there are often no symptoms, or there may be slight headache of a different character, or definite neuralgia, or some other functional disturbance.

It is not uncommon, as already mentioned, for some change in the character of the disease to occur at a certain period. Sensory disturbance may cease, or, if previously present, may come on. Thus one patient had hemianopia with the attacks of migraine until the age of fifty, when the visual disturbance ceased, and the headaches occurred alone. Occasionally, some morbid influence, chronic ill-health, acute disease, anxiety, or injury, may induce an increase in the intensity of the affection or a change in its character. Thus one patient, whose mother was the subject of migraine, suffered from simple "sick headaches" since childhood. At twenty-five he had a slight concussion of the head, falling against a wall. Hemianopia came on in a few minutes, followed by headache more severe than the patient had had before, and from that time each attack was preceded by the same visual symptom.

Migraine does not itself involve any danger to life. We have seen, however, that the vascular disturbance may lead in time to local vascular degeneration, and this affords an explanation of the observed fact that sufferers from migraine occasionally suffer from vascular lesions of the brain comparatively early in the degenerative period of life; but this sequel is rare. It may, however, occur in the part of the brain that has been the seat of the periodical functional derangement. Hemianopia habitually preceded the headaches in a woman who, after reaching the degenerative period, found one day that the hemianopia persisted after the pain. It was permanent, and due, as was subsequently ascertained, to a lesion in the opposite cuneus.* In some cases, after many attacks attended by intellectual impairment, some failure of mental power has been observed in the intervals.

Complications and Associations.—Vertigo is occasionally met with in the subjects of migraine, not only as part of an attack, but also as an occasional symptom at other times. Some of those who present it are in the second half of life, and the associated symptoms show that the vertigo is of the labyrinthine variety. Thus one patient, forty-seven years of age and gouty, who had been liable for many years to migraine, had an attack of vertigo with brief tinnitus and sickness; the watch was almost inaudible through the bone on each side, and no note of Galton's whistle could be heard through the air. In him tinnitus was inconstant, but heard occasionally on each side. It is probable that, in such cases, the central tendency to functional derangement renders the patient peculiarly susceptible to the influence of labyrinthine changes. I have several other examples of the same combination. In other cases, again, sudden attacks of vertigo occur without any indication of aural changes, and sometimes seem to be the result simply of the central instability. Thus one patient was liable, in the intervals between the attacks of migraine, to sudden sensations of unsteadiness, vaguely referred to the legs, without any aural symptoms. Another patient had sudden attacks, in which there was a tendency to fall backwards, accompanied by sickness. At other times she had paroxysmal headaches without sickness.

The relationship of migraine to other diseases is of great importance. That to gout has been already mentioned in the account of the causal relations of the disease. An alternation is often observed with some other form of neurosis, or at least a transition from one to the other. Migraine occasionally ceases, and is replaced by simple neuralgia. Many other instances of such transition have been collected by Liveing, as, for instance, to gastralgia, laryngeal spasm, anginal seizures, and paroxysmal insanity. In one case acute mania came on.

The most important, and one of the most frequent, of these associations is the relation of migraine to epilepsy. The connection of the diseases is of special interest because the sensory disturbance of the two has so many common features. I have met with many cases in which these maladies occurred in the same individual, a few of which have been mentioned in the chapter on "Epilepsy." In some instances migraine had existed for many years, and the patient afterwards became epileptic, the migraine usually ceasing or becoming much slighter when the epilepsy developed; but one epileptic began to suffer from migraine when his fits ceased. A recurrence of previous migraine on the cessation of epilepsy occurred in some cases. In a few cases both maladies co-existed in considerable intensity. In almost all the individuals who had suffered from the two diseases, the attacks of migraine were attended by well-marked sensory disturbance in addition to the headache and vomiting, and in one or two, abortive attacks of sensory disturbance occasionally occurred. In several cases, moreover, the epileptic attacks began by a local aura in the limbs, a rare feature in idiopathic epilepsy. In one instance of this, the first convulsive attack occurred after a fright. I have met with cases, in which epilepsy succeeded migraine and the epileptic fits seemed, as it were, to grow out of the attacks of migraine, being preceded by such sensory symptoms as had occurred before the attacks of headache. Thus a young man, whose sister was epileptic, began to suffer from attacks in which he saw glimmering lights in the right side of the field of vision, lasting about twenty minutes, and followed by headache, sometimes for half an hour, sometimes for the rest of the day, accompanied by nausea but no vomiting. He had such an attack every few months; one day a similar light appeared, brighter than usual, and after it had lasted for twenty minutes he lost consciousness in a convulsive attack, which, from the intensity of the subconjunctival ecchymoses, must have been severe. In rare cases of epilepsy, again, a visual aura may consist of fortification-spectra with colours, and even, as I have known, with hemianopia. In one such case the visual disturbance lasted ten minutes, occurring sometimes alone, sometimes with transient loss of consciousness, sometimes with a convulsive attack. Again, a woman had suffered from epileptic fits for two years, and also, since youth, from attacks of severe headache, lasting all day; in the course of the headache she would have attacks which began with a sensation at the epigastrium, passing up to the head; it seemed to spread over the head, and then the sight became dim, and this was followed by complete word-deafness and aphasia; after about ten minutes these symptoms suddenly ceased. Many of her epileptic fits were preceded by the same sensation starting from the epigastrium and going to the head.

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* Noyes, 'Journ. Nerv. Ment. Dis.,' 1889.

PATHOLOGY.—No anatomical changes are known to underlie the phenomena of migraine, and from the character of the symptoms, and the analogies of the disease, it is unlikely that any will be discovered. Hence the nature of the malady is a matter of inference, and hypotheses are sufficiently abundant and precise.

Two chief theories have been held regarding the origin of the attacks. One is based upon the alteration in the state of the vessels that is so conspicuous in the aspect of the patient. The pallor of the surface must be due to contraction of the arteries, the flushing of the skin to their dilatation, and it is assumed that a corresponding condition of the vessels of the brain is the cause of the derangement of function. The suggestion that spasm of the cerebral arteries is the cause of the symptoms was first made by Whytt, and the evidence in favour of this opinion, afforded by the state of the accessible vessels, was pointed out by Du Bois Reymond. Mollendörf urged that vascular dilatation, rather than spasm, caused the symptoms, and the fact that the condition varies in different cases has led to the theory, extensively held in Germany (by Eulenburg and most other writers), that there are the two varieties of the disease already mentioned, the "sympathetico-tonic" and the "sympathetico-paralytic" form, as they are sometimes termed. Dr. Latham, of Cambridge, who arrived independently at the conclusion that the attacks depend on vaso-motor derangement, has suggested that the early symptoms of the paroxysm are due to spasm, and the headache to dilatation of the vessels. According to these theories, the malady is essentially one of the sympathetic nerves.

According to the other, and alternative explanation of the disease, the primary derangement is of the nerve-cells of the brain. Their function from time to time is disturbed in a peculiar manner, and the visible vaso-motor disturbance is of secondary origin. The periodical derangement of function has been called, by a somewhat inapt metaphor, a "nerve-storm." This theory has been put forward and ably advocated by Liveing.

The sensory symptoms must depend on deranged action of the sensory centres in some part of the brain. They indicate a combination of arrest of action and of over-action in the nerve-cells concerned. In the language of modern pathology, there is a combination of inhibition and discharge; the loss of sight, for instance, must be due to inhibitory arrest of action, the visual spectrum to discharge. We have already seen (p. 739) that the same combination occurs in some attacks of epilepsy. The peculiarities in the disturbance of migraine are its special and often uniform features, deliberate course, and its limitation to sensory structures. To explain them on the vaso-motor hypothesis, we must assume, first, an initial spasm of the arteries in a small region of the brain; secondly, that the contraction always begins at the same place; and thirdly, that it can give rise to a definite, uniform, and very peculiar disturbance of function. There is no evidence of the truth of any one of these assumptions. As was pointed out in the discussion of the pathology of epilepsy, we are not justified in assuming that the state of the surface vessels and accesssible arteries is an indication of the condition of those of internal organs. If it were, inasmuch as the recognisable vaso-motor spasm is bilateral in almost all cases, even when the sensory disturbance is unilateral, we must assume a general contraction of the vessels of the brain. A general contraction could only cause a local disturbance of function by virtue of a local change in the functional tendency of the nerve-cells. But if such local change is admitted, the need for the vaso-motor mechanism disappears. Lastly, that vaso-motor spasm can cause a deliberate, uniform, and peculiar "discharge," is not only unproved, but in the highest degree improbable. In short, the difficulties in accepting the vaso-motor explanation of the sensory symptoms are so great that it could only be admitted as a tenable hypothesis if there were no other explanation of the coincidence of the two phenomena. But we know that the vascular system is in a special way under the influence of the cerebral centres. An emotional blush and the pallor of fear are conspicuous examples of this fact. It is at least as easy to conceive that the vascular changes are the result of the disturbance in the nerve-elements, as it is to regard them as its cause. The vaso-motor nerves are peculiarly sensitive to sensory impressions that are felt as pain. No symptoms at all resembling those of migraine have been observed when the sympathetic is distinctly diseased or deranged. We have proof, in the symptoms, of the deranged action of sensory cells; we know that cells are susceptible of primary disturbance of function, and there is at present nothing to justify us in going beyond this derangement in our search for the primary morbid process. The peculiar character of the visual impressions affords strong reason for regarding it as the result of a tendency to functional derangement in the cells themselves. This opinion is supported by the fact that it may occur as an isolated symptom, and that it may be related to an actual visual impression, as is shown by the facts mentioned on p. 846. Such an occurrence can have been due only to a primary functional disturbance of the cells themselves, and that which we feel sure exists in one case, probably exists in all.

What part of the brain is concerned in the production of the initial symptoms? Loss of speech must be due to disturbed function of the cortex. The sensory symptom in the limbs is like that which, in far more rapid evolution, precedes convulsions from cortical disease, and this source is therefore probable. The hemianopia also is best explained by the assumption of deranged function in the occipital lobe, especially since right hemianopia may correspond to almost simultaneous aphasia, and the opinion is supported by the fact mentioned above, that hemianopia was fixed by a lesion in the cuneus.

The cause of the headache is obscure. We know very little of the mechanism of this symptom in any condition. But when the pain is opposite in side to the sensory symptoms, we are obliged to assume that its seat is the cerebral hemisphere that is deranged; and the same conclusion is suggested when the pain begins on the side of the sensory disturbance, at some spot which has no definite relation to nerve distribution, as the very common initial seat, a small area in the temple. This is best understood by assuming a derangement of the sensory centres of the hemisphere which would receive impressions from this part. It is difficult to separate such pain from the symptoms that preceded it; the aspect of the patient is unchanged during its development. Flushing, heat, throbbing, come on subsequently, often as the pain spreads in area. How far its increase in extent and changes in character are due to a vaso-motor mechanism permitting arterial distension, we cannot say. Such a state may be secondary to over-action of the sensory centres, and may induce the spread of the disturbance. We must not ascribe too much significance to throbbing, or to the increase in the pain by the causes of vascular distension; these may be due merely to the over-sensitiveness of the central structures.

There is evidence, in many cases, of deranged function of lower sensory centres, those directly connected with the peripheral fibres. These are apparently the common seat of the disturbance in neuralgia, and we have seen that cases of migraine which begin with distinct cerebral symptoms may be followed by pain, more or less definitely neuralgic in character—even tenderness of the skin, vaso-motor disturbance, and (although exceptionally) an eruptive state, or actual herpes zoster. But it is possible that the lower centres may be affected secondarily to the higher. We must not assume that influences cannot traverse nerve-fibres in the direction opposite to that in which these habitually conduct, for we have an instance of this in the propagation of trophic influences down the peripheral sensory nerves. But only when such symptoms exist, or pain corresponds in area to nerve distribution, are we justified in assuming that the sensory centres in the pons and medulla are specially disturbed. Some other considerations regarding headache are discussed in the next chapter. When all has been said that can be, mystery still envelops the mechanism of migraine.

The nausea and vomiting throw no distinct light on the processes of the attack. We have seen, in many affections, how wide-spread is the central representation of the pneumogastric, and that vomiting may result from organic disease in any part of the brain. In migraine it seems as though the nervous discharge of the act of vomiting afforded some relief to a morbid state of the nerve-cells, but only when the disturbance has nearly run its course and is ready to subside.

The hypothesis that the derangement is primarily one of nerve-cells of the brain enables us better to understand the relation to other neuroses, and especially that to epilepsy, which is occasionally so distinct. In epilepsy, as we have seen, we must assume a disturbance of function, in some cases so similar in character that we cannot doubt the identity of its seat with that of migraine. But the process in the two differs in its course, associations, and other features, and these imply an essential difference in its minuter characters. These relations, however, make it intelligible that the two should occur in the same subject, and that intermediate forms of nerve disturbance should sometimes be met with.

Diagnosis.—From other forms of headache, that of typical migraine is distinguished by its sensory accompaniments, and, if these are absent, by its paroxysmal character, severity, definite course, and in many cases, as its name suggests, the unilateral distribution of the pain. But it is important to remember that no line of distinction can be drawn between simple paroxysmal headaches and migraine. Every intermediate form is met with, and "sick headache" especially is always migraine. In malarial headaches, the intermissions are shorter and the cause is generally traceable. The pain of chronic brain disease is more or less constant; it may present remissions and even intermissions, but not the longer intervals of migraine. The sensory disturbance in the limbs, and the affection of speech, may excite a suspicion of acute brain disease, but it is only in a first attack of migraine that the question will arise, and even then the brief duration of the symptoms, and the peculiar features of the visual disturbance, sufficiently indicate the nature of the attack. When these occur without headache, a mistake is common. Such patients are often thought to have grave organic disease. But a knowledge of the occasional isolation of the sensory symptoms, the history of preceding attacks, and of some pain, which can usually be obtained, almost always enables a correct opinion to be formed.

Another difficulty is presented by the diagnosis from minor epilepsy, in which the visual aura may closely resemble that of migraine. In most cases of epilepsy with a visual aura, this is brief, lasting only a few seconds, while the visual disturbance in migraine lasts twenty minutes to half an hour. The fortification-spectrum is strongly suggestive of migraine rather than epilepsy, although not quite conclusive. The occurrence of a convulsion, of course, decides the question, but where the patient's account is all we have to guide us, we must make our diagnosis from the general features of the case, not forgetting that the one disease may unquestionably pass into the other, and that some attacks seem to be of intermediate nature.

A diagnostic difficulty of another kind arises in cases in which the subjects of migraine become affected with some other malady, and the symptoms of the former continue, and complicate the latter. Thus one patient was attacked with Bright's disease; he had retinal changes and optic neuritis, and the intensity of the headache led to a diagnosis of intra-cranial tumour. But the pain was purely paroxysmal, and of the same character as that to which he had been long liable. The opinion that there was no organic cerebral disease was confirmed after death. In another case, a subject of migraine became affected with general paralysis of the insane, and the sensory disturbance that had preceded the attacks of headache occurred alone, and complicated the paroxysmal symptoms of the cerebral disease.

PROGNOSIS.—The prospect of recovery from migraine, that is of the entire cessation of the attacks, is never considerable, but in most cases a diminution in the frequency and severity of the attacks, often reducing them to a trivial and undisturbing degree, may be anticipated from persevering treatment. The prognosis is best when the malady is of short duration, and there is some removable condition in the general health or mode of life which has distinctly contributed to the production of the malady. The longer the disease has lasted the less is the prospect of a cure, but neither this nor hereditary tendency has an unfavorable influence on the prognosis equal to that of conditions which we cannot yet discern, and which may render one of two apparently similar cases quite intractable, while the other yields at once and permanently. In the second half of life the tendency to cessation is greater, provided the malady has not recently developed. The prognosis is better when the face is at first pale and pinched than when it is at first flushed. It is better when there is a family history of gout than when other neuroses can be traced

TREATMENT.—If any error in mode of life, or defect in general health can be traced, the removal of these is the first and most essential step in treatment. The details vary in every case, but the most frequent conditions that need attention have been already indicated in the account of the causes of the disease. Of especial importance are increased rest, regularity in meals, attention to diet; whatever is known to induce a paroxysm should be carefully avoided. Hot, crowded rooms are especially injurious. If inherited gout is probable, the regimen suitable for this should be adopted.

The special treatment consists, first, in the continuous administration of drugs during the intervals, with the object of rendering the attacks less frequent and less severe; and secondly, the amelioration of the attacks themselves. As a rule, the measures that do good when employed during the intervals have no influence on the attacks.

There is much variation in the effect of the intermediate treatment. As already mentioned, the measures that do great good in one case will fail in another, apparently quite similar. The influence of bromide in epilepsy naturally leads us to turn first to this as likely to be of service in a malady that has so many features in common with that disease. In some cases it is of service, but less often than might be expected. It is most likely to be efficacious in the cases in which there is no change in the colour of the face, or in which the face is flushed throughout an attack. Ergot may often, then, be usefully combined with it. In the majority of cases, however, and especially in those in which there is conspicuous pallor in the early stage, the drug that has most influence is nitro-glycerine. Given regularly during the intervals, just as bromide is given for epilepsy, it has a striking effect in many patients, rendering attacks far slighter and far less frequent, and often, after a time, stopping them altogether. It should be given twice or three times a day, after food. If taken when the stomach is empty, it passes rapidly into the blood, and may cause brief cephalic discomfort, which though not objectionable in itself, sometimes deters the patient from continuing the medicine.

To avoid causing alarm, it is therefore also desirable to begin with a small dose, $\frac{1}{150}$, $\frac{1}{180}$, or even $\frac{1}{200}$ of a grain. The most convenient mode of administration is in the 1 per cent. solution in alcohol, now termed Tinct. Trinitrini (B. P. Sup.); it can be given in combination with tinctures or acids, but is decomposed by alkalies. A very useful combination is with tincture of nux vomica, tincture of gelsemium, and dilute phosphoric acid, or with citrate of lithia and the acid syrup of lemons. Sometimes more benefit is derived from the combination of this with bromide, when a little hydrobromic acid should be added instead of the phosphoric acid. If there is much dyspepsia, it may be given with hydrochloric acid, and some liquid preparation of pepsine. The trisnitrate of bismuth may also be given with it. I have found such combinations of the liquid preparation of nitro-glycerine with other drugs far more useful than the administration of nitro-glycerine in tablets. It is not well to continue it during an attack; at the very onset a dose may be taken, but if this is not effective the medicine should be omitted till the attack is over; it seldom gives relief to the symptoms, and occasionally makes them worse. It is possible that the drug acts chiefly by periodically flushing the nerve-centres with arterial blood, and so improving the nutrition and the function of the nerve-cells. A purgative at the onset will occasionally cut an attack short in some patients, but more often it fails.

During the attack, absolute rest is essential, and is indeed impressed on the patient by the distress occasioned by activity. The recumbent posture is generally that in which the sufferer is most comfortable. All strong sensory impressions should be avoided. Although a dose of alcohol or of nitro-glycerine will occasionally cut short a commencing attack, when the headache is developed, it is usually increased by drugs that dilate the vessels or excite the heart. Relief is afforded to the pain by a full dose (thirty, forty, or sixty grains) of bromide, and its effect is increased by the addition of five or ten minims of tincture of Indian hemp; this may be repeated every two or three hours. But more often relief is obtained from antipyrin, acetanilide, exalgin, or phenacetin. Unfortunately their influence is apt, after a time, to cease; a change from one to another sometimes renews the benefit. Twenty grains of chloral, or fifteen of butyl hydrate (croton-chloral) usually makes the patient drowsy and easier for a time, but after an hour or two the pain returns in its former severity, and the duration of the attack does not seem to be lessened. The same is true of most other sedatives. A hypodermic injection of morphia often acts no better than other sedatives, but occasionally it does give great relief, and may terminate an attack that has already nearly run its course. When there is much mental depression during the attack, valerian and assafœtida have been found useful by Latham. Drugs that cause contraction of the arteries are almost powerless; all that a full dose of ergotin does is to lessen the throbbing intensification of the pain complained of by some patients. Strong tea and coffee are popular remedies, and occasionally give some distinct relief, which may also be obtained from a few grains of caffeine. Guarana (the powdered seeds of *Paullinia sorbilis*) has been often used. It contains an active principle, guaranine, which is identical with caffeine.* Three or four doses of guarana, twenty or thirty grains, may be taken at intervals of half an hour, or a single dose of sixty grains, or guaranine may be given in doses of one to five grains. It has for the most part disappointed the expectations that were raised by the praises at first bestowed upon it. Some relief is often given to the pain, but the relief is transient only, and it is doubtful whether guarana is really more effective than tea and coffee.

Local applications sometimes afford relief—sedative liniments of belladonna, aconite, &c., or simple counter-irritation by a mustard plaster to the nape of the neck. From the last, some patients experience relief more uniformly than from any other measure. Solid menthol is occasionally useful, rubbed on the skin for a few minutes where the pain is greatest, but it is too feeble an agent to have much influence on any severe pain. Sometimes relief is afforded by a hot bath of mustard and water to the feet, and in all cases it is well to keep the extremities warm.

Electricity is of little service. Faradism usually does harm. The voltaic current passed through the head occasionally gives transient, but rarely permanent or considerable relief. Repeated galvanisation of the sympathetic has been recommended as a remedial measure, one pole pressed deeply in front of the sterno-mastoid, the other held in the hand, and it is advised that when the symptoms of vaso-motor spasm predominate, the positive pole should be placed over the sympathetic, and the negative when there is evidence of vaso-motor paralysis (Berger, Horst). The value of the treatment is, to say the least, seldom perceptible.

HEADACHE.

Headache is a symptom of almost all kinds of morbid states of the system, and cannot often be regarded as, in itself, an independent disease. Nevertheless it is a symptom for which patients seek treatment more often than for any other of equally varied causation. Hence it may be useful to indicate the chief varieties that are met with, and briefly to describe the symptom in its apparently primary form. As an indication of organic disease it has been already described.

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* Guarana contains twice as much of the alkaloid as tea, and five times as much as coffee.

We know almost nothing of the structures in which the pain of headache is felt or the mechanism of its production (see p. 97). The subject is one on which it is easy to theorise, but there are no facts that give to any hypothesis a considerable degree of probability. One conclusion is, however, suggested by the symptoms,—that the seat of the pain varies in different cases. Outside the skull are nerves that frequently give rise to pain, but all pain that corresponds to the course or distribution of nerve-trunks must be regarded as neuralgic, and not as headache. Some diffuse headaches seem superficial in character, but they may still be due to processes, not in the nerves, or the grey matter in which the nerves directly end (which is supposed to be deranged in neuralgia), but in higher centres of the cerebral hemispheres through which superficial pain is perceived. This is especially probable when the pain is local, but does not correspond to a nerve area, resembling in this some forms of migraine. This feature is, however, more frequent in occasional exacerbations than in the continuous pain. More frequently headache seems to be deeply seated, and the pain is referred to structures within the cranium. Of such structures the membranes, when inflamed, are the seat of intense pain, which is probably really produced in them, because other organic disease, such as a tumour, which is seated at the surface of the brain and involves the membranes, may also cause intense pain corresponding in locality to the disease. Whether pain is produced directly by morbid states of the cerebral tissue we cannot say. We are certainly not justified, however, in denying the possibility of this seat of pain (see p. 97). It may be caused by organic processes which do not involve the surface. After necrotic softening in the central ganglia, headache is common on the side opposite to the hemiplegia, and the diffuse pain of "hemicrania" may be on the side opposite to that of the sensory symptoms.

When pain is felt, in whatever part the nerve-processes occur that cause it, the consciousness of pain is related to the activity of certain nerve-cells of the cortex of the brain. In pain that is produced in the limbs, for instance, the cortical cells concerned are those that are related to, and receive impressions from, the fibres by which the sensory impulses pass from the periphery. A similar relation must exist in the case of sensory impressions from the head itself—from the structures covering the head, from the bone, and probably from the membranes. The pain of meningitis, for instance, if due to the irritation of the meningeal nerves, must be perceived by means of the activity of those cells in the cortex that receive the impressions from the membranes. Whether there is a similar representation, in the cortical cells, of the tissue of the brain itself, we do not know. Sympathetic nerve-fibres accompany the arteries in the cerebral substance; there is reason to believe that the functional state of the cortex influences the state of its arteries (as is the case in all other organs), and this must mean a relation of the cells of the cortex to the vaso-motor centre; hence it is quite possible that there may be a sensory representation of the substance of the brain, at least of its interstitial tissue, in the cortex of the brain itself. The phenomena of epilepsy seem to show that the central representation of the head, and perhaps of the brain, is connected in a special manner with the structures related to the mental state of consciousness. It is very common for an aura, felt first in the epigastrium or limbs, to seem to ascend to the head, and when it reaches the head, consciousness is almost invariably lost. The aura is the result of the spread of the discharge in the brain, and the phenomena indicate that the central sensory representation of the head has a special importance, and it may be that it has a special susceptibility. But a functional derangement of the cortical cells in which the membranes are represented would, by itself, explain many forms of headache.

It may be held, indeed, that the primary derangement in headache is not of the highest sensory cells, but of the cells of lower centres, to which, for instance, the nerves of the membranes directly pass, but this view is less easy to reconcile with the difference between headaches and the true cranial neuralgias (in which the lower cells are probably concerned), and also with the resemblance often observed between the headache of organic and of functional origin. When pain is felt in the side of the head on which the hemisphere is deranged or diseased, its origin in lower centres can be certainly excluded; and the same inference (of cerebral origin) is probable when severe pain on the opposite side is associated with other symptoms, and is felt only in a part of a nerve area.

Another mysterious factor in the mechanism of headache is the influence of the vessels. It is probable that mere vascular dilatation, passive or active, will cause pain;* it is certain that pain already existing is increased by even trifling mechanical congestion. We do not know how the vascular dilatation causes pain. There are no facts to show that the nerves of the vessels ever become sensitive. It is certain, however, that the intra-cranial pressure must be increased by the dilatation, since, however quickly the movement of the cerebro-spinal fluid may compensate for vascular repletion, this movement is due to the mechanical pressure from the vessels, and this pressure must act on the whole brain. We know how sensitive nerve-fibres are to pressure; it is highly probable that their sensitiveness is vastly exceeded by that of nerve-cells. If the sensory cells of the cortex, in which the cranial and intra-cranial sensitive structures are represented, are the most readily influenced of all the sensory cells, we can understand that headache should result from vascular repletion.

The frequency with which pain due to general causes is referred to the frontal region is very peculiar, and does not seem to be susceptible of any satisfactory explanation. It is occasionally a feature of the pain of organic disease, such as a tumour, situated away from the frontal region. Diffuse pain is not often referred to this region alone in primary simple headaches, although pain in other parts of the fifth nerve, as that of dental irritation, may spread up to this region. In simple headache, the pain may seem to pass through the head from the back to the front, or *vice versâ*, or may be referred to the supra-orbital regions or the temples.

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* It is certain that pain results from mechanical congestion, but we cannot separate the effects of the state of the vessels and the increased amount of venous blood in the brain.

Varieties.—Toxæmic headache may be the effect of either acute or chronic blood-states. Of the acute form, the typical example is that of fever, in which the pain is generally frontal, rarely occipital or general, and scarcely ever vertical or one-sided. In slight cases it may have a neuralgic character for a short time, and may be felt in one part of the head, as the temple, but this form is usually transient. The pain is commonly dull in character, but often very severe, and is rendered worse by stooping or by whatever causes mechanical congestion of the head. The deep-seated character of the pain suggests that it is referred to some intra-cranial structure. There is no evidence that it depends on any vascular state; the increase by mechanical congestion is a common feature of almost all severe headaches.

Many poisonous substances cause headache as one of their toxic effects, especially when the dose is small, or as an after-effect of large doses. Some of these poisons, such as nitrite of amyl and nitro-glycerine, dilate the vessels, and the pain may be in part or altogether the effect of the active congestion produced; but it is also possible that the pain is sometimes the direct effect of the agent on the nerve-elements. The inhalation of a small quantity of ether, as, for instance, when the air of a room is charged with it in moderate degree, generally causes headache, which seems to be the direct effect of the ether, since it is not attended by any sign of vascular distension.

Chronic toxæmic states often cause very severe headache, which may be quite intractable unless its cause is discovered. In the morbid blood-state that is caused by kidney disease, for instance, there is often intense headache, usually frontal and continuous, but with occasional exacerbations. It is sometimes increased by alcohol in a special manner, but this is a feature also of some other forms. The severity of the pain has many times given rise to an erroneous diagnosis of cerebral tumour in cases in which there is albuminuric optic neuritis. Headache also results from diabetes, from chronic alcoholism, lead poisoning, and from similar toxæmic states.

Congestive headache is often produced by mechanical hindrance to the return of blood from the head. It is generally frontal, but sometimes general. A tight collar, for instance, or rest with the neck so bent as to compress the veins, frequently causes slight headache of this character. The repeated mechanical congestion produced by cough is another frequent cause, and may even occasion the patient more distress than the cough itself. Mechanical congestion not only distends the veins, but causes over-filling of the capillaries, and increases the amount of venous blood in the brain. Thus there are several factors at work to which the pain may be due. Active congestion of every kind also gives rise to pain very similar to that produced by passive hyperæmia, but more distinctly throbbing in character. The action of poisons that dilate the vessels has been already mentioned.

Gastric disturbance is another exceedingly common cause. The pain is most frequently occipital or vertical; now and then it is frontal. Sometimes there is slight pain of a superficial character; the severe neuralgic headaches associated with vomiting cannot, as we have seen, be ascribed to gastric influences. We do not know by what mechanism stomach disorder causes headache—whether it is by some influence on the cerebral vessels, or whether by absorption of some toxic material from the stomach or bowel,—some morbid secretion, or product of the imperfect digestion of food, or altered bile. A purgative often relieves the pain, and old theories regarded the bile as an influential agent in the production of this and other symptoms; but when there is much bile circulating in the blood, as in jaundice, headache is usually trifling or absent.

The term "neuralgic headache" may be applied to the forms in which the pain seems to be superficial, in part or altogether, and is limited in area, but does not correspond to the course or distribution of the superficial nerve; such pain is one of the most common varieties of headache. The subjects of neuralgia often suffer from continuous or occasional pain in the head, which may be felt in any part, sometimes general, but more often local, and not distinctly paroxysmal. It may seem to arise by an extension of the pain of true nerve origin in an adjacent region. Such local headaches are common in hysteria. Many forms, such as the sensation of a nail being driven into the vertex, are intermediate between neuralgia and headache. A common cause of supra-orbital pain is the use of the eyes when there is hypermetropia. The strain on the ciliary muscle seems to be the immediate excitant; the pain goes off when the use of the eyes for near objects is discontinued.

Anæmic headache is met with both in the common form of anæmia, in which there is chiefly a deficiency of hæmoglobin (as in chlorosis), and also in cases in which much blood has been lost. The pain is frontal or general; in chlorosis it is sometimes peculiarly intense at the back of the eyes, and is accompanied by a sensation as if the eyes were being dragged back into the head.* Neuralgic headache and true neuralgias of all kinds are also common under the same conditions.

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* The fact that optic neuritis may result from anæmia is of great interest in connection with the occurrence of headache, since it shows that the disturbance of the nerve-elements may be more than functional, and suggests that their nutrition may be impaired in cases of apparently functional disturbance. It is noteworthy that in Bright's disease, when optic neuritis preponderates over retinal changes, headache is often especially severe.

Brain-work and brain exhaustion are other common causes: sometimes there has been an amount of brain-work that is absolutely excessive; in other cases the amount has not been large, but is excessive on account of the deficient nerve-strength of the individual. Pain from this cause is often general, sometimes local, and then generally felt in the forehead. Occasionally a very intense form of headache results, especially where there has been an acute breakdown from overwork. It may even be so severe as to cause a suspicion of meningitis.

In various conditions of nervous weakness—"neurasthenia," as it is now termed—headache is common, and may have almost any seat or character. Many cases in young girls are of this character, and are often associated with symptoms of hysteria; the headache is then called hysterical, but it is doubtful whether there is any closer association with that disease than the state of defective nerve-strength which underlies both conditions. Indeed, by those who adopt the prevalent custom of using the term "neurasthenia" in its widest application, almost every form of headache, except the toxæmic, may be ascribed to this cause. Severe continuous headache is sometimes met with in the early stage of constitutional syphilis, but chiefly in those who have recently undergone a long course of treatment for the disease. This also has been regarded as a manifestation of "neurasthenia syphilitica." Many of the slighter continuous headaches associated with nervous weakness are largely maintained by attention, as is the case in even greater degree with the cephalic sensations presently to be described.

Diagnosis.—The diagnosis of the form and nature of headache consists first in the search for any general condition on which it may depend. It is important to note the time at which the pain comes on, its relation to food, to mental work, and to other influences that may possibly be concerned in its causation. The general state of the patient must also be carefully investigated, and the effect of various influences in augmenting or reducing the pain. Anæmic headaches, for instance, are often relieved by the recumbent posture; those of congestive or toxæmic origin are especially increased by stooping or effort or mechanical congestion, but not always by simple recumbency, which, perhaps through the rest it involves, often lessens the pain, and does so also in cases of neuralgic nature. The seat of the pain is often suggestive, but too much weight must not be placed on this indication, since many variations are met with. The pain produced by acute blood-states, such as fever, is generally frontal and deeply seated, and is increased by cough and movement. The forehead is also a frequent seat of neuralgic headaches; these are, however, generally superficial in character, and are often felt just over the eyebrows. Pain of gastric origin is also sometimes frontal. Pain in one or both temples is generally paroxysmal and allied to migraine; so are all one-sided headaches. Occipital pain, deeply seated, felt constantly or daily, is more often due to disturbance of the stomach than to any other cause. Vertical headache is seldom due to blood-states; occasionally it also is of gastric origin, but when very limited it is usually allied to neuralgia. General headache may be of almost any nature; it is often due to toxæmic conditions, anæmia, and conditions of nervous weakness. It must be remembered that blood-states which, on account of their slight degree or other causes, do not produce considerable headache, sometimes cause slight local pains.

TREATMENT.—The most important element in the treatment of headache is the discovery and removal, as far as possible, of the state of the blood or nervous system on which it ultimately or immediately depends. The measures necessary for this object must vary with each variety of headache, and cannot here be even enumerated. Attempts to relieve the headache by direct treatment usually fail unless the cause is discovered and removed. Careful inquiry may be needed to discover its real origin. Thus a patient came for treatment for headache, in whom it was caused solely by cough, the result of phthisis. It is important that in all cases except those in which there is evidence of anæmia, the patient should sleep with the head well raised, and with the shoulders raised as well as the head, so that there is no compression of the cervical veins by flexion of the neck. Most forms of headache, except those due to anæmia or simple weakness, are relieved by aperients, which probably act partly by lessening the amount of blood in the cephalic vessels, and partly by improving the state of the blood. Diuretics are useful in many toxæmic headaches. The treatment of the superficial and very limited varieties is similar to that of migraine. Indeed, most of the advice given respecting the treatment of the headache of migraine is applicable also to its more simple form.

Headache of every kind is often relieved, although seldom removed, by external applications that sting the skin. Chloroform, or any stimulating liniment, will answer the purpose, provided evaporation is prevented by an impermeable tissue. Bisulphide of carbon has been recommended for the purpose, but it does not seem to have any special value. Mustard plasters to the back of the head or neck are useful when the pain is occipital, and often induce beneficial sleep. Slight pain about the forehead may be removed by the application of menthol, which is especially useful when such pain is just enough to prevent sleep.

Sedatives are very uncertain in their influence. Opium and morphia are seldom useful, and often do more harm than good, partly in consequence of the indirect effect of the constipation that is produced. Gelsemium and Indian hemp frequently lessen the pain; the former chiefly in superficial forms about the front of the head connected with neuralgia, the latter not only in neuralgic, but in anæmic, and also other ill-defined forms of headache. Bromide of potassium may be given in addition to the Indian hemp if there is restlessness, irritability, or insomnia, but has not alone much influence on the pain. Butyl-chloral is occasionally serviceable. Antipyrin, acetanilide, and phenacetin often give great relief, especially to paroxysmal exacerbations of pain, but are apt soon to lose their power. By varying them, however, their influence may often be maintained. It must be remembered, however, that these palliative measures ought to be regarded only as adjuncts to the causal treatment.

HEAD PRESSURE AND OTHER CEPHALIC SENSATIONS.

It is common for patients to suffer from various more or less distressing sensations in the head, which have not the character of actual pain. These sensations are much more frequent in men than in women. They occur chiefly during the first half of adult life, between twenty and forty, but are sometimes complained of by lads of sixteen or eighteen, and occasionlly by persons in the second half of life, at fifty or sixty. In late life they are relatively more common in females, coming on sometimes after the cessation of the menses, at the "climacteric period." They are sometimes associated with a gouty state of the system. Brain-workers are chiefly liable to these sensations; they are seldom met with among those who earn their living by manual labour, and hence are rarely complained of by hospital patients. Many of the sufferers are the subjects of pronounced hypochondriasis, who attend to their sensations, and often complain of other kinds of nerve disturbance. Excessive brain-work may be the distinct exciting cause of the symptoms; in other cases they succeed a definite headache, of short or long duration, severe or slight. I have known the sensation to follow a blow on the top of the head. It is indeed probable that, in a large number of cases, they are set up by some real sensation, sometimes of pain, sometimes of other character. A distressing sense of constriction over the whole scalp succeeded, in one case, the application of an ice-cap for a few days, on account of delirium during influenza.

The sensations vary in different cases, but by far the most frequent is a sense of pressure, sometimes trifling, sometimes as if a heavy weight were on the head, and occasionally as if the top of the skull were being driven in. Many patients describe the sensations in language that is clearly exaggerated, so that it may not be easy to ascertain the actual intensity of the sensation, but it is certainly often very distressing. It may be called "headache" or "pain," but however intense it is, inquiry shows that the sensation has not the character of actual pain, either acute or dull. Its common seat is the vertex and top of the head, but it is sometimes felt at the back of the head, less commonly in the forehead. One patient described the sensation as a feeling as if there were an iron band round the back of the head. Sometimes it is lateral, in the parietal region or the temples, "as if the sides of the head were being driven in." Much less common is the opposite sensation, a feeling of expansion, as if the bone of the skull were being pushed out or up. A sensation of heat or burning, not amounting to pain, is sometimes described. A vague sense of fulness is not uncommon, and it is sometimes accompanied by a sense of throbbing; in other cases, again, the feeling is said to be as if the skull were empty. Various other sensations are occasionally described, which the patient, in default of adequate terms, describes by some simile beyond the range of ordinary experience, such as "a feeling as if the brains were being stirred up with a stick," or "as if red-hot coals had been placed on the brain," or "as if the head were being alternately opened and shut." Occasionally a superficial sensation, of tingling, creeping, &c., is complained of in some part of the head, on one side or both.

The sensation is often uniform in the same case, and may continue unchanged for many years. Less commonly the patient has first one sensation and then another. The sense of pressure may seem, when most intense, to give rise to dull, actual pain. The discomfort is commonly increased by brain-work, and often by any unpleasant emotion, while it may be unnoticed when the mind is occupied by some agreeable subject. It is often only observed when the patient is indoors, being unfelt when he is out in the open air.

The chief agency in the production of these sensations is certainly the mental state of the patient. There is probably at the outset some actual sensory impression, often some headache, and the attention is constantly directed to the part, with the result that the patient perceives sensations which, under normal circumstances, would be unperceived. Nerve-impulses, in health unnoticed, must be continually passing from all parts to the centres, and they may be readily perceived if attention is directed to them. A person imagines that he is unconscious of his body and limbs, but let him direct his attention to any part, and in a few moments he will be conscious of a distinct sensation in it, and with especial readiness of sensations in the head. If the reader will direct his attention to the vertex, he will probably soon be able to detect a distinct sense of pressure there, especially if he is fatigued or has been engaged in mental work. It is easy to understand that such a sensation may be increased by constant attention to an unpleasant and even distressing degree, especially if some real sensation originally "drew" the attention to the part, and if the individual is in feeble health, or of a nervous temperament. The physical side of "attention" is a lowering of resistance in nerve-cells, so that they are more rapidly roused to activity, and their activity is greater than normal. Thus the ultimate result may be the same as if the afferent nerve-impulses were increased in energy, and a sensation is produced by nerve-processes that should be unperceived. The condition may be described as a "receptive dysæsthesia."

Rarely, when the sensation is at the worst, there is slight tenderness of the scalp. Although the sensations are usually constant, in exceptional cases they are paroxysmal, and then may be accompanied by some general nervous excitement and insomnia. It is common for the patient to say that sleep does not refresh him. There is occasionally also a little mental confusion. An inability to concentrate the mind on a particular subject, with a little consequent failure of memory, is often described, but is merely the result of the engrossing sensation which obtrudes itself on the mind and prevents attention. The sensations are sometimes distinctly increased by stimulants. Very rarely there is slight flushing in the face during paroxysmal sensations, but this is not common.

Some of these sufferers complain at times of other slight functional nerve disturbance. An actual headache may alternate with the sense of pressure, &c. In other cases there are occasional sensations of giddiness, almost always vague and slight, and perhaps sometimes scarcely real, since the patient looks out for them. Often it is merely a sensation of the "legs giving way."

The treatment of this condition is a matter of very great difficulty. Sedative drugs have little influence upon it, unless they are given in large doses, and then their effect is transient. This fact is in harmony with what has been said of the probable pathology of the affection. As long as the influence of attention is at work, the patient's distress continues, and the mental cause is beyond the influence of drugs. The sufferer cannot, by a deliberate effort of the will, ignore the sensations that distress him. The only method of treatment that is effective is to make the patient realise the unimportant nature of the sensations, and try to neglect them by directing his attention to other subjects. While it is wise for him to avoid whatever greatly increases the sensations, he should persistently endeavour to "snub" them, rather than to cultivate them. When this effort is made and maintained, the discomfort slowly ceases to trouble the patient, and at last is no longer noticed. Unfortunately, the mental constitution of some patients makes it impossible for them thus to ignore their suffering; they continue to attend to the sensations, doubt the assurances of physicians who assert the unimportant nature of the discomfort, and seek the advice of others, placing most trust in those who pander to their apprehensions by the assurance that the sensations indicate some grave disease, such as congestion of the brain. Many a patient of this class may be truly said to be suffering from a *morbus medicorum*, and his best chance of recovery is to keep away from doctors altogether. Of course any conspicuous defect in general health should be put right; a gouty diathesis should be treated; distinct nerve-weakness may require tonics; and if there is vertigo or insomnia, bromide, with small doses of Indian hemp, gives some relief.

FACIAL HEMIATROPHY.

Facial hemiatrophy, as its name implies, is a wasting of one side of the face, in which, however, the bones and subcutaneous tissue suffer more than the muscles. The precise relations of the disease are uncertain. It is commonly classed with exophthalmic goitre and migraine, but it is very doubtful whether this is its true position. Many facts connect it with organic disease of the fifth nerve, but the relation between the two is not yet ascertained with sufficient uniformity and definiteness to justify its inclusion among the effects of disease of that nerve. In many cases it has followed a blow or contusion of the face, sufficiently severe to have acted on the bone, generally in either early or later childhood. Sometimes the atrophy has developed in the course of a year or two, and it has generally affected chiefly the upper and lower jaws. Instances are—a blow on the angle of the orbit; contusion of the upper and lower jaw in a child of three, whose head was jammed and twisted between iron railings; a blow and exposure to cold at twelve, the atrophy being considerable at fourteen. In other cases, some trifling local disorder has been the apparent cause, as the forced passage of a sound down the lacrymal duct at sixty-three, an "abscess in the ear," and "erysipelas of the face."[*] An instance of the affection was first recorded by Parry in 1825, but the disease only received attention after it had been described by Romberg thirty years later. It is very rare, and is of small practical importance; it involves no danger to life or health, and cannot be influenced by treatment.

Causes.—The disease generally begins in early life, sometimes in childhood, sometimes in the second decade. It has commenced as early as two or three years of age. It occasionally develops in adults, rarely in late life. A disparity in size between the two sides of the face is sometimes congenital, but such cases are commonly distinguished from those that commence after birth. Females seem more liable to the affection than males. Most of the cases have been isolated, but in one recorded case an aunt of the patient had suffered from the same disease. It has often come on without any exciting cause, but in some cases, the affection has followed a morbid process, local or general, of which it seemed to be the consequence. Examples of local influences have been already mentioned, and, in addition, the atrophy has supervened on some disease of the fifth nerve, such as neuritis, either primary (Mendel), or secondary to some other disease, a tumour beside the pons, or an inflammation of the upper jaw bone, spreading to the nerve. Among the cases after general disease is one in which the affection commenced on the right side of the face after an attack of scarlatina with diphtheria at six, and an attack of typhoid many years afterwards was followed by distinct atrophy of the left side. It has also followed "typhus" (probably in most cases typhoid), at five and at eighteen. Other general diseases which have preceded it, as acute rheumatism and influenza, may have acted by setting up changes in the fifth nerve or its nucleus, for in many instances early pain in the corresponding side of the head suggested such a process.*

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[*] A blow, Wolff, 'Virchow's Archiv,' Bd. xciv; Stewart, 'Montreal Med. Journ.,' 1889; contusion, Skyrme, 'Brit. Med. Journ.,' March, 1892; inflammation of the bone, Ruhemann; passage of a sound, Borgherini, 'La Psich,' viii; abscess of the ear, Preobashenski, 1890; erysipelas (at nine), Cohen.

SYMPTOMS.—A gradual diminution in the bulk of one side of the face has often been the first indication of the disease. In some, however, the atrophy has begun at one spot, commonly on the cheek, at which the skin has been observed to become thin and pale from loss of pigment, and depressed from the loss of the subcutaneous tissue. The hairs drop out in the affected parts and the sebaceous follicles waste. The condition has gradually spread, and has involved the bones. In the cases that began in early life, the ultimate difference between the two sides of the face has been greater than in those that developed later, a hindrance to growth increasing the effect of the wasting, so that it is chiefly in these cases that the disparity in the size of the bones is considerable. In these cases also the usual growth of hair does not take place on the face, although there may not be much difference in the anterior part of the scalp. The ultimate condition in well-marked cases is very striking. The appearance of the patient is as if the face were made up of two halves from different individuals. One side may have the fulness of youth, and the other the furrows of age, while the contrast in character is intensified by the difference in size. On the affected side the bone of the forehead and cheek is considerably smaller than on the other side; the skin covers the bone closely, and so presents depressions without counterparts on the opposite half, while a vertical furrow usually marks the transition from the affected to the normal side. The side of the lower jaw may be only two thirds the length of its fellow, and is diminished in depth, so that the face appears much shorter. The cartilage of the nose shares the wasting, and in the cases that begin early, it may be so small on the affected side as to render the nose altogether unsymmetrical, the tip deviating towards the smaller side; the external ear has been known to share the wasting; the teeth are sometimes small, and may drop out or decay early. In spite of the wasting of other tissues, the muscles seldom seem to be altered, either in size, in voluntary power, or electrical excitability; rarely they have been wasted.

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* Typhus or typhoid, Rona, 1889; Dreyer, 1890; influenza, Jolly, 1891.

Indications of implication of the fifth nerve are sometimes distinct. Sensation may be impaired or lost, either in all parts or in the second and third divisions, and taste may be lost on the front of the tongue. Rarely there has been some wasting of the muscles of mastication on the affected side, and they have been the seat of spasm in three instances,* tonic (in the case due to a blow), and paroxysmal tonic and clonic contractions, which afterwards spread to the tongue, in the case recorded by Sachs. In a few instances the corresponding halves of the tongue and of the palate have also been wasted. It is doubtful whether there is any alteration in the size of the arteries and veins. The ultimate wasting of the skin may be very great; it is said that a fold of the skin on the affected side may be only one tenth the thickness of a similar fold on the other side. In several instances, the wasting was preceded by scleroderma.† The temperature is usually unchanged, but in the early stage it has been found to be slightly lessened. The organs of special sense on that side are not changed, but the eye often appears retracted in the orbit in consequence of some wasting of the orbital tissue. Neuro-paralytic ophthalmia has been once met with (Graff). The secretion of saliva and of tears is not affected. As a rule there is no wasting beyond the region of the fifth nerve, but a case of bilateral atrophy has been already mentioned, and Virchow described a typical hemiatrophy of the face, associated with an atrophy of the skin and subcutaneous tissue of part of the back and arm on the same side; in the arm, in the back, it extended from the fourth to the seventh dorsal spine, it affected chiefly the radial side of the forearm. This case has become important on account of its pathological investigation by Mendel. A curious case of local wasting of all the subcutaneous tissues in small areas on the back and arm has been mentioned in vol. i, p. 481.

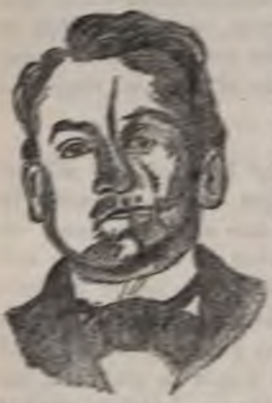

FIG. 158.—Facial hemiatrophy (Schwann).

The affection, beginning commonly in childhood, progresses steadily for some years, and then becomes stationary and remains so for the rest of life. The first illustration (Fig. 158) is the case of a man named Schwann, who exhibited himself in this and other countries a few years ago; he was then aged forty; the atrophy began in childhood, and his case was described by Romberg in 1851. The other figure is of a child of seven in whom the affection showed itself at three and half, six months after an accident in which the face was jammed and the neck twisted. The child was subject to attacks of migraine.

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* Wolff, Muratow, Sachs.

† Gibney, Rosenthal, Muratow, Nixon. In the case of the two latter, scleroderma existed elsewhere.

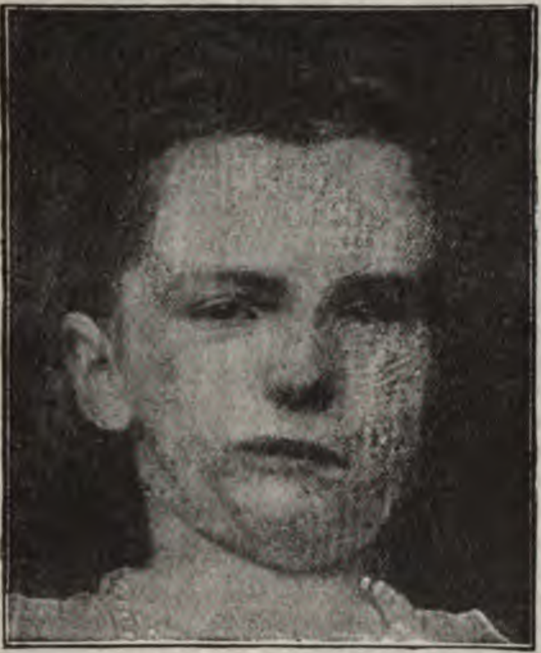

FIG. 159.—Hemiatrophy of the face in a child of seven.*

Facial hemiatrophy seldom co-exists with other maladies except the occasional affection of the fifth nerve. In the above case there was also a liability to attacks of migraine, in another there were symptoms of tabes (Jolly), while in one remarkable case the condition co-existed with multiple exostoses of the bones of the head and face.†

PATHOLOGY.—The great variation in the conditions under which, and causes from which, facial hemiatrophy seems to arise, makes it difficult to ascribe all cases to the same mechanism, and yet other features make it almost equally difficult to separate the immediate causes.

Especially the correspondence of the hemiatrophy, in all cases, to part or the whole of the region supplied by the fifth nerve, is a fact of clear, although limited significance, which is increased by the occasional evidence of impaired function of the nerve (corresponding to the atrophy in a case in which the area of the first division was unaffected), and is made still more definite by certain recent pathological observations. In Virchow's case, Mendel found, seven years later, interstitial neuritis of all parts of the nerve, with degeneration of the fibres, and atrophy of many in the upper root and of the cells of the locus cæruleus. (In the cord slight changes were found in the anterior cornu corresponding to the wasting in the back.) Hence he conjectures that the upper root of the fifth contains trophic fibres. The tumour beside the pons, in the case already referred to, had compressed the fifth nerve and the pons (as well as the nerves of the eyeball). There was conspicuous narrowing of the fibres of both lower and upper roots of the fifth; the cells of the locus cæruleus were normal. Moreover, facial hemiatrophy has been produced in dogs by dividing the fifth nerve within the skull (Girard). It thus seems certain that disease of this nerve may cause the affection, but, on the other hand, the effect is not a common consequence of such disease, even when complete and of long duration, and in many cases the only evidence of such a relation is correspondence in distribution or some early pain in the affected parts. Hence it has been thought to be due also to other causes. Wasting of the corresponding side of the face has been described by Seeligmüller in a case of injury to the cervical sympathetic, but has been absent in most cases of the kind.* A subsidiary mechanism—compression of the arteries for the bone by the cutaneous and subcutaneous changes—has been suggested, but is a far-fetched hypothesis.

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* I am indebted for this illustration to Mr. Skryme, who has published the case ('Brit. Med. Journ.,' 1892, p. 654).

† Karewski, 'Dieut. med. Zeit.,' 1891.

It is important to remember the fact that the nutrition of all parts, except the muscles, seems to depend on the posterior root-fibres, to which the fifth nerve chiefly belongs, and that the influence of chronic lesions is to cause a slow wasting distinct from the acute disturbance produced by irritation (see vol. i, p. 22). The atrophy of the skin and bone in the finger ends, after a lesion of the nerves of the arm, may proceed, in spite of normal sensibility, and is a state closely allied to facial hemiatrophy.† The evidence of a trophic influence does not, however, afford any proof of trophic nerves. The rapid conduction of sensory impulses upwards may be compatible with a slower conveyance of a trophic influence downwards, even in the same fibres, and the latter may be lost while the former is unchanged. There is not sufficient evidence to justify the assumption that the mechanism is a damage to fibres of the sympathetic that run in the fifth nerve; although implication of these fibres may have co-existed in the rare cases with evidence of disturbance of the sympathetic.

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* Much weight has been placed on a case described by Brunner, in which the condition was associated with prominence of the eyeball and pallor of the skin, supposed to be dependent on an affection of the sympathetic. But the case stands almost alone.

† Even thickening of the skin may occur in neuritis, as sometimes in the face, cf. vol. i, p. 68.

The chief indication of the facts is that there is a loss of a special influence exerted through the fibres of the fifth nerve, independent of its conducting functions, although sometimes impaired together with them by organic disease. It is an influence such as we can trace elsewhere, not only in the conditions mentioned, but in the trophic changes in tabes. To what origin of the fifth, it is related we can only learn definitely by further investigations, but its loss may not be necessarily associated with visible alterations in the nerve-elements on which it depends.

It may be instructive, in this connection, to remember that arthritic muscular atrophy may be set up by merely transient joint pain, and that it is not attended by any evidence of organic changes in the motor nerves, through which it is certainly produced. Being extensive, but limited in range and often persistent in some degree, it is thus a process bearing some analogy to facial hemiatrophy, especially to such cases as that produced by the forced catheterisation of the lachrymal duct and those due to transient injury. Both diseases and the trifling nature of many of the apparent causes of facial hemiatrophy, show how little ground we have as yet on which to erect theories of the causation of this malady.

Diagnosis.—The only condition with which the affection can be confounded is that in which there is a congenital difference between the two sides of the face. In this, however, the inequality is slight, and on the smaller side there is not the difference in the growth of the hair or the colour of the skin seen in most cases of hemiatrophy.

Prognosis and Treatment.—There is no prospect of improvement, except, possibly, in some of the rare cases that come on in adult life after injury, or are accompanied by distinct symptoms of disease of the fifth nerve. The treatment of these is that of the morbid process in the nerve. If improvement ever occurs, it is probably consequent on the removal of the cause.

EXOPHTHALMIC GOITRE.

Exophthalmic goitre is a mysterious malady, usually manifested by three sets of symptoms: enlargement of the thyroid, prominence of the eyes, and a peculiar disturbance of the vascular system, viz. over-action of the heart and increased pulsation of the arteries of the neck. These vary in relative degree in different cases, and any one of them may be inconspicuous. The disease was first fully described by Graves in 1835,* who drew especial attention to the coincidence of over-action of the heart and enlargement of the thyroid, and described the co-existence, with these, of prominence of the eyes, in a case communicated to him by Stokes. He thus described all the symptoms, duly recognising the predominance of the cardiac disturbance. In Germany the first full account of the affection was given, in 1840, by Basedow. Hence the affection is often called in this country and in France "Graves' disease," but in Germany "Basedow's disease."* Many other designations have been proposed—"struma exophthalmica," "cardio-thyroid exophthalmos," &c., no one of which has obtained currency or can claim special exactness.

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* Cases had been published earlier, but without recognition of all the symptoms or their special character; in 1802 by Flagani; in 1816 in the 'Medico-Chirurgical

While the three symptoms named above are usually present, and have hence been termed the "cardinal symptoms" of the disease, any one of them may be absent. Moreover, any of them may be met with in association with the less common symptoms, or complications of the disease. Hence, while the majority of the cases present definite and characteristic features, cases occur, more or less closely allied to the typical form, but extremely various in their characters. It is not practicable (nor would it be useful) to do more than indicate the occurrence of these, in describing the leading features of the malady.

Causes.—Women are far more prone to suffer than men, the ratio being uncertain, but probably about twenty to one.† The disease may occur at any age, from two to sixty, but is met with chiefly in adult life, between fifteen and fifty, and more cases begin between twenty and thirty than in any other decade. I have known it to commence at fifty-one and fifty-three, and also at seven and a half in an epileptic girl, whose mother suffered from it from eighteen to twenty-five. Although not usually inherited, instances such as that just mentioned are occasionally met with. In several recorded instances two sisters, or mother and daughter, have suffered; in one a mother and a son aged eight, in another an aunt and three nieces (Cheadle). Eight out of ten children suffered in various degrees and at various ages in a family observed by Oesterreicher.‡ In such instances, some of the cases were slight or imperfect, so that they

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Journal,' anonymously; in 1818 by Demours (thyroid and eyes only); by Parry in 1825 (posthumous publication of a case observed in 1785); and by Adelmann in 1828. See Wickham Legg, "Note on the History of Exophthalmic Goitre," 'St. Barth. Hosp. Rep.,' vol. xviii, p. 7; and Maitland Ramsay, 'Glasgow Thesis,' 1891, which contains the best account of the history of the disease.

* This term is firmly rooted in Germany, but if the disease had now to be named, it is probable that the Germans, ever ready to give credit where it is due, would term the malady "Graves' disease." It has been also called "Parry's Disease."

† Recorded series have presented singular discrepancies, apart from those that are due to the field for observation. In some the proportion has been about 5 to 1, but Russell Reynolds met with only one male in a series of forty-nine cases, 'Lancet,' May 17th, 1890.

‡ 'Wien. med. Presse,' 1884.

would probably have escaped recognition had not attention been directed to them by the other cases. Much more frequent is general neuropathic inheritance, indicated by insanity or epilepsy in relations. Thus, I had at one time under my care two sisters, one for epilepsy, the other for severe exophthalmic goitre; another patient had a father and an uncle epileptic, another a sister and an aunt epileptic. A family relation to diabetes has also been noted.* The causes of ordinary goitre seem to have little influence in producing the exophthalmic form,† and recovery has actually followed removal to a place where goitre was endemic (Trousseau). In a very few cases, however, the special symptoms of the disease have supervened on goitre of the common character and of some years' duration.

General debility and anæmia are unquestionably powerful predisponents. In many cases, the first symptoms have shown themselves after a period of constitutional weakness due to some distinct and adequate cause, such as pregnancy, exhausting discharges, especially hæmorrhage, weakness after childbirth or abortion, &c. Amenorrhœa sometimes precedes the onset. But the disease may come on in a person who otherwise appears to be in perfect health. In some instances, organic heart disease has preceded the malady, but the association of the two is not frequent, and its significance is uncertain.

No immediate cause is so frequent as emotion,—sudden terror, or prolonged distress. The symptoms have been known to follow emotion with great rapidity, and to become distinct after a few days. Many cases were seen in Alsace and Lorraine after the Franco-German war. The quick action of the heart, due to alarm, may persist as that of exophthalmic goitre. A fright at nine caused tremor, which persisted, accompanied by stammering, and during the next eight years the symptoms of exophthalmic goitre gradually developed.‡ Many apparent causes have involved alarm. In one case it followed the removal of both ovaries. Violent exertion has also, in some instances, appeared to set up the derangement—mountain climbing, severe vomiting, or violent cough. Among other rare causes are stimulating climatic influences, injuries to the head, cold,§ alcoholic excess, various acute diseases, scarlet fever, pneumonia, rheumatism, and diarrhœa. It is probable that in most of these a predisposition existed, for in many other cases, no exciting influences can be traced.‖

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* Manby, 'Brit. Med. Journal,' i, 1889 (three instances).

† In India it is said that, although endemic goitre is common, the exophthalmic form is practically never seen among the natives (Brockman, 'Trans. Oph. Soc.,' 1866, p. 117).

‡ Lewin, 'Inaug. Diss.,' Berlin, 1888.

§ See, for instance, a case recorded by Dr. Bristowe, 'Trans. Oph. Soc.,' 1886, p. 46.

‖ Russell Reynolds (loc. cit.) observed an unusual number of cases during the cold sunless weather of 1888.

SYMPTOMS.—The onset of the disease is usually slow and gradual. In rare cases it is acute, and these may run a rapid course and end in death, or the acute onset subsides into a chronic stage. Occasionally one or two slight attacks, perhaps recognised only afterwards as of similar nature, occur and pass away, months or years before the onset of graver symptoms.

The three sets of symptoms in eyes, thyroid, and heart, seldom commence together. As a rule the cardiac disturbance leads the way. Palpitation troubles the patient for months or even years before the other symptoms are added. The enlargement of the thyroid and prominence of the eyeballs often come on together; if one develops first it is usually the former. These three leading symptoms we may first consider in detail, but two others, a hindrance to the descent of the upper lid on looking down, and a peculiar tremor of the limbs, are so frequent (being present in more than half the cases) that some observers have thought that they should be classed with the three leading symptoms just mentioned.

Heart.—The characteristic condition of the heart is excited action, shown by increased frequency and increased force of its contractions. The degree of excitement varies from time to time, and is readily increased by emotion or exertion. At first, between the exacerbations, the action may be normal, but gradually the subsidence becomes less complete and the over-action persistent, only varied by attacks of more violent "palpitation." The increased rate of action is sometimes trifling, and the pulse does not exceed 90—100 per minute. More often the rate is higher, 120 or 140, even when tranquil; sometimes 150 or 160, and under excitement it may rise to 180 or 200. It is usually regular. The impulse is unduly sharp, and felt over a larger region than in health. The action of the heart resembles that of a person under considerable nervous excitement or after exertion. The area of cardiac dulness is sometimes normal when the area of impulse is increased, but usually, after a time, there are signs of enlargement of the heart. The impulse extends far outwards and becomes diffused, while the cardiac dulness is also increased to the left. These signs of dilatation usually preponderate over any indications of hypertrophy. The dilatation is secondary in time, and doubtless also in origin, to the excitation of the heart. The sounds are usually increased in loudness; but the first sound is often short.* Occasionally they are accompanied only by the murmurish roughness that is common in excited hearts; more often, however, there is a distinct systolic murmur, which may be loud. It is heard over the whole heart, loudest near the sternum or at the base, but also audible at the apex, and sometimes outside the apex. There is probably then actual mitral regurgitation, the result of the dilatation which the mitral orifice suffers with the ventricle. Slight mitral endocarditis has been occasionally found after death, and is perhaps due to the mechanical strain on the valves. The impulse of the heart may be attended by a thrill. As the disease advances, the attacks of palpitation increase, and are often accompanied by considerable dyspnœa, which is, indeed, sometimes an early symptom.

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* Graves mentioned a case in which the sounds were audible four feet from the patient.

The *arteries*, especially those of the neck and thyroid, present increased pulsation and are dilated. Sometimes the abdominal aorta presents a similar pulsation, but it is only in rare cases that the arteries of the limbs share the derangement. Indeed, the radial pulse, though of course unduly frequent, is usually smaller than normal, and presents a marked contrast to the strong pulsation of the carotids. A systolic murmur, often loud, may almost always be heard in the arteries of the neck and thyroid, continuous with that heard at the base of the heart and over the aorta. Sometimes it is to be heard in other arteries, even in the femorals. A venous pulse has occasionally been observed in the neck, and even in the arm, probably due to the influence of the pulsation of an artery on the flow through an adjacent venous trunk. The vibrations in the carotid are often so coarse as to be felt as a thrill.

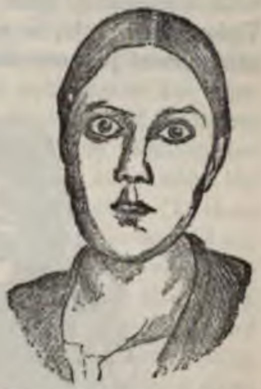
FIG. 160. — Exophthalmic goitre; early stage. Slight enlargement of the thyroid; considerable prominence of the eyes.

The *thyroid* usually enlarges after the cardiac symptoms have existed for some time. This "struma" as it is sometimes termed, is a slow, painless enlargement, at first soft, uniform, but sometimes greater in one lobe than in the other, and occasionally confined to one lobe or to this and the isthmus. When one lobe is the larger, it is rather more often the right than the left; very rarely only the isthmus has been enlarged. When the organ is increased in size, the arteries of the thyroid always pulsate strongly; sometimes the whole gland seems to pulsate; a distinct arterial thrill may be felt, and a loud blowing systolic murmur may be heard, rarely musical, still more rarely double. The superficial veins over it may be enlarged. The degree of enlargement is not often extreme; seldom equal to that which is common in ordinary bronchocele. It varies somewhat in size from time to time, being greater, as Graves noted, during the attacks of palpitation. Diminution in size is often accompanied by an increase in firmness. In many cases the enlargement is only trifling in degree, and it is occasionally absent altogether.

Eyes.—The prominence of the eyes, like the enlargement of the thyroid (with which it often coincides), commonly succeeds the cardiac over-action. In rare cases it has been the first symptom. It may be equal in the two eyes, or occur first and remain greater in one eye.

It is very rarely confined to one side; then the thyroid is said to be most enlarged on that side, but exceptions to this rule are common; I have twice known the eye and thyroid to be affected on opposite sides.* In degree it may be so slight as to easily escape notice, or merely to cause a "staring" appearance, but it is often so considerable that when the patient looks straight forwards the white sclerotic is visible above and below the cornea. It may be extreme in degree, so that the insertions of the recti are exposed, the movement of the eyes limited, and so that the lids cannot meet over it, even in sleep. It is said that the eyeball may even be dislocated from the socket and may have to be replaced with the finger. The degree of prominence varies from time to time with the over-action of the heart. When it is greatest, pain may be felt in the eyeball. There is also, in some cases, an actual increase in the size of the globe, especially in the antero-posterior diameter, which has been found to be one tenth greater than normal (Neumann). Extreme prominence may interfere with movement, apart from the paralysis to be described later. Vision, as a rule, is unaffected, but myopia may result from the enlargement just mentioned. The pupils are almost always normal;

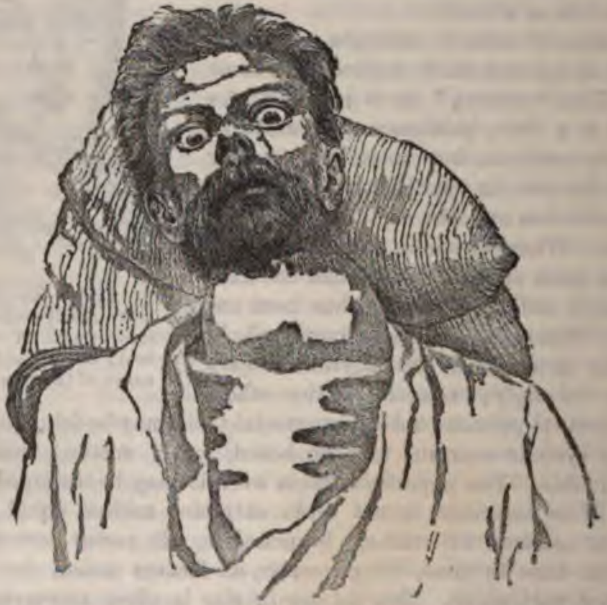

FIG. 161.—Exophthalmic goitre. Defective descent of the upper lid on looking down. (After Wilks.)

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* A remarkable case has been recorded by Burney Yeo, in which great enlargement of the right half of the thyroid coincided with prominence of the left eye, and when right exophthalmos came on, the left lobe of the thyroid enlarged ('Brit. Med. Journ.,' 1887, i, 320).

inequality and slight irregularity have very rarely been met with. When the lids fail to cover the eyes, these are often dry in the morning. Corneal inflammation is sometimes met with, apparently due to the imperfect protection of the globe when the lids fail to meet; conjunctivitis is not rare, and occasionally there has been opacity of the cornea, and even sloughing, generally in both eyes, but in one before the other. Œdema of the lids is occasionally present; I have seen it considerable in degree, and associated with œdema of the conjunctiva, in a case in which the prominence was slight. The ophthalmoscopic appearances are usually quite normal, with the exception of the occasional pulsation of the retinal arteries and veins. The latter is due to the effect on the vein of the pulse in the adjacent artery within the nerve; when the latter is increased the vein is more easily influenced, and the venous pulse may appear, although the vessels are not quite near enough for a normal degree of pulsation of the artery to act on the vein. Hence the conditions for this communicated pulsation are more frequent in this disease than in health, just as they also are in aortic regurgitation.* Œdema of the disc is occasionally met with, slight in degree; its occurrence is not related to œdema outside the eyeball. Atrophy of the optic nerve has been observed (Emmert), and is probably due to the stretching of the nerve.

Another frequent ocular symptom has been already mentioned—the defect in the descent of the upper lid that should occur when the eyeball is rotated downwards. It is met with in two forms—(1) there is no descent or only a very trifling movement; the lid is held up, sometimes even more than in health, by a "retraction" of the lid (Figs. 161 and 163); sometimes it exists on one side only (Fig. 162). (2) The lid does descend, but is retarded, lags behind the globe, and descends in an hesitating, halting manner, now suddenly falling a little, then stopping, and again moving down suddenly for a short distance, the movement of the globe meanwhile being uniform. It may at last descend as low as the prominence of the globe permits (which is generally a little above the level of the canthus) or may stop short of this point. In the latter case the upward movement, of necessity, does not quite correspond with that of the globe, but when the lid begins to rise its ascent is uniform. It is important not to mistake for a retarded descent the mere effect of the prominence of the eyeball, which may render impossible the proper approximation of the lids. The defect in descent is limited to the movement of the lid with the globe, and does not occur when the lids are closed by a contraction of the orbicularis, as in going to sleep. It may present every variation in degree, so that there is no real distinction between retraction and hindered descent. The latter is often called v. Graefe's symptom, the former Stellwag's, but v. Graefe's description included retraction,† and Stellwag only described increased width of the palpebral fissure (an obvious consequence of the defective descent), and added nothing to our knowledge of the phenomenon. It may occur early, when the exophthalmos is trifling, or be absent when this is extreme. Rarely a similar defect in the ascent of the lower lid has been noted. Its possible causation will be considered presently.

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* See 'Medical Ophthalmoscopy,' 3rd ed., p. 20.

† 'Berlin med. Gesellsch.,' 1864, and 'Deutsche Klinik,' ib., p. 158. He stated

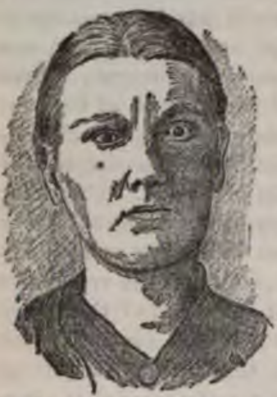

FIG. 162. — Exophthalmic goitre, early stage. Retraction of the left upper lid.

Retraction of the lower lid has been very rarely observed.[*] Occasionally there is some weakness of the ocular muscles, especially of the internal recti, lessening the power of convergence,[†] and more extensive ophthalmoplegia has been observed (see p. 882). The pupils are almost always normal; very rarely there has been undue contraction or dilatation.

Of the three chief symptoms of the disease, not one can be said to be invariable. Prominence of the eyes is absent in at least one tenth of the cases, goitre in perhaps one twelfth. The cardiac disturbance is almost constant. It is apparently absent in rare cases, but, on the other hand, is often alone obtrusive. Occasionally the heart is not enlarged, although its action is quick; there may also be severe paroxysms of palpitation and dyspnœa. Increased pulsation of the arteries of the neck is very seldom absent, and a careful examination often detects a slight enlargement of the thyroid and slight prominence of the eyes, when these are not obtrusive. When the latter does not exceed the degree that is met with as a normal condition, the friends of the patient often recognise it as unnatural. If preponderant cardiac disturbance makes rapid progress, the patient may die before other symptoms attain such a degree as to attract attention. When the enlargement of the thyroid and exophthalmos precede the

that the associated descent of the lid on movement of the eye was "almost completely arrested or reduced to a minimum," sometimes even when the prominence was slight. Stellwag v. Carion ('Med. Jahrb.,' 1869, Heft ii, p. 25) described the increased width of the fissure, the lessened frequency of involuntary winking, and the defective correspondence in movement of the lid and globe. V. Graefe only referred to the descent of the lid, and most writers have followed him, limiting the symptom to this by implication. A few have (it would seem erroneously) included impaired correspondence in ascent among the features alluded to by v. Graefe. The limitation of the defect to descent, which most writers have tacitly implied, has been formally emphasised by Bruns ('Neur. Centralbl.,' January, 1892). "Unnatural elevation of the lid" was first mentioned by White Cooper ('Lancet,' 1849, quoted by Ramsay).

[*] By Dr. Hill Griffith, 'Trans. Oph. Soc.,' 1886, p. 61.

[†] See Möbius, 'Deutsch. Zeitsch. f. Nervenh.,' 1891, where a full abstract of recent observations will also be found.

derangement of the heart, they increase very rapidly after the latter has developed.

The general health of these patients usually suffers. Anæmia is frequent, although not invariable. Menstruation is irregular and often ceases. Loss of flesh occurs, but is less marked in the face than elsewhere. Pyrexia is frequent in the later stages of the disease, usually moderate, from 1° to 3° F., but it is inconstant; in many cases the temperature is perfectly normal throughout, while in others it is elevated at an early stage, either for short periods or persistently. The pyrexia has occasionally been such as to suggest tuberculosis or typhoid. It has been noted that the urine does not then present the characters of ordinary febrile urine. There may be a subjective sense of heat, without actual fever. Profuse perspiration, sometimes of a penetrating odour, is an occasional symptom, and is especially common in acute cases. It has been confined to the side on which the thyroid and eye were alone enlarged (Lewin). The "tache cerebrale" is often to be obtained. The appetite is frequently deficient, sometimes capricious.

Other symptoms on the part of the nervous system may be present. A sense of throbbing in the head, and a pulsating noise in the ears, are occasionally complained of. Headache is sometimes troublesome, and may be one-sided. There is often insomnia, and sometimes transient vertigo. Mental depression or irritability is very frequent; the latter, indeed, is almost a characteristic feature.

Another very common symptom is muscular tremor. It is conspicuous in at least half the cases, and may be discerned, in slight degree, in most. It varies in character, but is usually a rather coarse, somewhat jerky tremor, occurring on movement only. In some cases it is so regular as to resemble that of paralysis agitans; in others it is so irregular as to remind the observer of chorea. When regular, it is a little more frequent in time than that of paralysis agitans.[*] It may be general or partial, and has been known to be unilateral when the goitre and exophthalmos were also unilateral, and, in a regular form, to be one of the first symptoms of the disease.[†]

Occasional Symptoms and Complications.—Many cases have been recorded in which there was also glycosuria. The change in the urine has followed in some cases, and preceded in others, the special symptoms. In one instance of this complication all the symptoms followed a blow on the head (Brunton). In another it succeeded an eruption of urticaria, but previously there had been frequent brief paroxysms of intense sense of heat of skin with palpitation (Budde). The sugar has been known to lessen when the other symptoms improved. Intermittent albuminuria has been recorded by Begbie, and since observed in many cases. Increased secretion of pale urine is

[*] According to Marie 8—9½ per second; paralysis agitans 5—6 per second; but, as we have seen, that of paralysis agitans varies much.

[†] Möbius, 1883.

common, and has been observed to precede paroxysms of palpitation and dyspnœa.

Enlargement of the lymphatic glands sometimes occurs in the course of the affection, resembling that of lymphadenoma, but moderate in degree, and not progressive. It may develop rapidly, and slowly subside. I have seen three very marked instances of this complication; one was a severe and fatal case of exophthalmic goitre in a girl whose sister suffered from lymphadenoma. Enlargement of the tracheal and bronchial glands has been observed by G. de Mussy.

The skin presents occasionally other morbid states besides those mentioned. Local redness, and even erythema, have been observed. A diminution in the electrical resistance is almost constant (Begbie, Vigoroux, Eulenberg, Wolfenden[*]). Instead of being equal to 4000—6000 ohms, it is reduced to 1500, 1000, 900, or even to 300 ohms, with an E. M. F. of 15 volts. It is ascribed to the dilatation of the blood-vessels and the moistening of the skin by perspiration. It has little practical value, for its determination causes considerable pain, and a similar reduction is met with in other diseases, as hemiplegia, epilepsy and chorea.[†]

Abnormal pigmentation of the skin is not uncommon in some degree, as vitiligo, or increased pigmentation in the same positions as in Addison's disease, except that it is very rarely met with in the mucous membrane of the mouth.[‡] It is sometimes associated with leucodermia. The pigmentation generally occurs after the other symptoms are fully developed, but has been known to precede them. It has also been known to pass away with the over-action of the heart, when the other symptoms became stationary.[§] Loss of hair has also been observed.[‖]

Slight subcutaneous anasarca is occasionally met with in anæmic subjects, and sometimes there has been more considerable œdema, peculiar in character or seat. In one case with glycosuria, terminal œdema increased from the feet to the umbilical level, and there stopped (Möbius). The peculiar subcutaneous fulness above the clavicles, common in neuralgia, &c., has also been met with (Rendu). It was accompanied by some pigmentation of the skin in another case (Sollier), and preceded the exophthalmos and cardiac over-action, the thyroid being small. I have observed myxœdematous swelling of the eyelids and pigmentation of the skin to follow permanent recovery from exophthalmic goitre. Myxœdematous symptoms have hardly ever been observed to coincide with enlargement of the thyroid; exceptions are—a case observed in Thibet,[*] and one in an epileptic woman (Kowalevski). Usually the thyroid has been atrophied; it was so in a case in which transient general œdema occurred on emotion and at the menstrual periods.[†]

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[*] See also Charcot, 'Gaz. des Hôp.,' 1885; Eulenberg, 'Berl. kl. Wochensch.,' 1889; Cardew, 'Lancet,' 1891.

[†] In chorea it has been found as low as 350 ohms. (Wolfenden, 'Practitioner,' 1887).

[‡] Where, however, it was found by Oppenheim, 'Berl. Gesselsch. f. Psy., &c.,' Dec., 1887. Several instances of pigmentation are described in a very instructive lecture by Drummond, 'Brit. Med. Journ.,' 1887. See also Carrington, 'Trans. Oph. Soc.,' 1886.

[§] Davies, 'Brit. Med. Journ.,' 1891.

[‖] Barnes, 'Brit. Med. Journ.,' 1889.

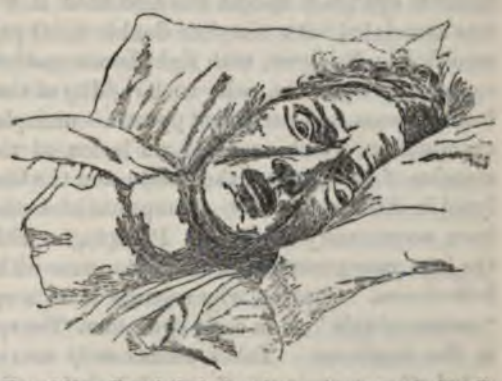

FIG. 163.—Exophthalmic goitre. Acute mania (fatal). (From a photograph.)

An important occasional complication is mental derangement. We have seen that slight mental change (irritability, restlessness, depression), is common; the derangement now referred to amounts to actual insanity, which often commences with hallucinations. Its form varies: it may be melancholia, or simple mania or recurrent mania, or even general paralysis of the insane.[‡] Mania is the most common condition, and may be the cause of death. Fig. 163 represents the aspect of a woman suffering from acute mania, which came on in the course of exopthalmic goitre and was fatal in a few weeks. Acute mania, indeed, generally leads to speedy death, but now and then subsides into chronic insanity. Hysterical symptoms are also common in this disease, and may present various forms, of which the most common is some variety of anæsthesia or paralysis. But symptoms of paralysis due to organic disease have often been ascribed to hysteria, in this malady.[§] Restriction of the fields of vision has been observed, and found to vary with the action of the heart.[‖]

The tremor already mentioned has amounted in some cases to a degree of clonic spasm or choreoid movement that must be regarded as a complication. In a few cases in childhood or youth, true chorea has been supposed to exist. In one instance this was probable, since the movements increased for a time, and then subsided. In the other cases, however, the spasmodic disturbance persisted, and its nature is therefore doubtful. Tetanoid spasm and painful cramps have been described.

Many other kinds of functional derangement of the nervous system, besides those already mentioned, have been observed to coexist with exophthalmic goitre. Epilepsy occasionally occurs, generally in subjects who possess an inherited tendency to it. Neuralgia, and migraine in typical form, have also been met with. Paralysis of cranial nerves sometimes develops, apparently due to an affection of their nuclei, although some degree of weakness of the ocular muscles may be the result of degeneration of their fibres. Besides the defect of convergence already mentioned, the whole of one third nerve has been paralysed, and in other cases the fourth or the sixth, while complete external ophthalmoplegia has also been met with. In one case this was associated with complete double facial palsy (Ballet) and in a case recorded by Bristowe, with right hemianæsthesia (including the special senses), epileptic fits, palsy with rigidity of the right side, hæmorrhages from the ears, and persistent pyrexia—unexplained by the post-mortem examination, which revealed no lesion of the nervous system. The muscles of mastication, the palate, and the tongue have also been paralysed in different cases.[*] Symptoms of disease of the spinal cord have been occasionally observed. Paraplegia, with flaccidity or rigidity of the legs, may come on; in the latter case with excessive knee-jerk and foot-clonus. Charcot, who regards it as a special form, has observed "astasia-abasia" as an early symptom. The sphincters may be involved in the weakness. Tabes occasionally coexists, but has usually preceded the symptoms of exophthalmic goitre. Chronic muscular atrophy has been observed.[†] Subacute atrophic paralysis in the arms was preceded, in one instance, by spasm, tonic and clonic, before the special symptoms of the disease came on,[‡] and in a case described by Drummond, the same disease was followed by acute myelitis.[§]

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[*] 'Brit. Med. Journ.,' 1891, p. 755.

[†] Sollier, 'Rev. de Méd.,' 1891.

[‡] See Savage, 'Guy's Hosp. Rep.,' 1882; Drummond, loc. cit.; Reynolds, loc. cit.; Schenk, Inaug. Diss., Berlin, 1890.

[§] *E.g.* Ballet, 'Soc. méd. des Hôp.,' 1888.

[‖] Kast and Willbrand, 1891.

Many symptoms occasionally met with are apparently due to vaso-motor disturbance. Sweating has been already mentioned; in a case recorded by Money a copious acid sweat broke out on the central parts of the face during attacks of palpitation and extreme frequency of the pulse.[‖] Diarrhœa is sometimes troublesome, and is thought to be a kind of intestinal sweating. Occurring early, it has been mistaken for sporadic cholera. Atony and distension of the colon, general or partial, may often be detected by percussion.[¶] Occasionally there is an abnormal flow of tears. Local œdema in the limbs, local congestion of surface vessels, atrophy of the mammæ and other parts, some skin diseases, and even gangrene, have all been regarded as manifestations of vaso-motor disease related in some obscure manner to the primary disorder.[**]

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[*] Féréol, Möbius, Jendrassik, Potain, &c.

[†] Cazal, 'Gaz. hebd.,' 1885.

[‡] Huber, 'Deut. med. Wochensch.,' 1888.

[§] Drummond, loc. cit.

[‖] 'Trans. Oph. Soc.,' 1886, p. 113. It is noteworthy that this patient had an epileptic aura consisting of "an awful smell that seemed to ascend from the left side of the body and reach the mouth," but it was not followed by any actual fit or loss of consciousness.

[¶] Federn, 'Berlin klin. Wochenschr.,' 1888.

[**] In the curious case recorded by Burney Yeo (p. 876, note), the occurrence of exophthalmos, first on one side and then on the other, was accompanied by loss of the hairs of the eyebrow and eyelid.

Course and Varieties.—The onset is usually gradual, and that of the cardiac over-action often cannot be fixed. The duration of the disease is generally prolonged through several years. But the differences in course are extreme. Occasionally the onset is acute; marked symptoms may develop in a few days, commonly after some psychical disturbance, and especially when there is an hereditary tendency to the disease. They may pass away in a week or two. Thus a boy, aged eight, the son of a woman who suffered from the disease, after a disappointment at school, was seized with palpitation and profuse sweating; the next day the thyroid was large, the eyes were prominent, and the pulse was 180. After two days more, the symptoms gradually disappeared, and in ten days he was well (Sobrig). Such acute cases may, however, run a rapidly progressive course, and end in death at the end of six weeks (Bamberger), but cases so rapid are extremely rare. Death at the end of six months is uncommon, and, on the other hand, in many cases the disease lasts for five or even ten years. An acute onset may subside into a chronic stage, similar to that of the cases that commence gradually.

Considerable and prolonged remissions are frequent, and sometimes actual intermissions occur, of such duration and completeness that the patient, in effect, suffers from several distinct attacks. I have known a patient to have three such attacks at intervals of several years. Pregnancy is sometimes accompanied by improvement. Permanent recovery may also occur. It is most frequent in the cases that develop rapidly, and in which the cardiac symptoms preponderate over those in the neck and eyes. What proportion of cases recover is uncertain. It has been estimated at a quarter, and this is probably not an over-statement, if the cases are included in which the cardiac symptoms are the chief. But a considerable degree of severity seems to preclude recovery. I have seen one patient in whom well-marked symptoms, great prominence of eyeballs and enlargement of thyroid, coming on in early womanhood, passed away entirely, and twenty years later there had been no recurrence.* Even when there is not complete recovery, however, the symptoms may subside from a most distressing degree to comparative insignificance. Just as the cardiac symptoms commence before the others, so they may persist long after the enlargement of the thyroid and prominence of the eyes have disappeared. The malady is said to run, on the whole, a more rapid course in men than in women.

In most fatal cases, death seems to be the result of the cardiac affection, and of increasing general weakness. Attacks of palpitation and dyspnœa become more frequent and troublesome; œdema of the legs comes on; exhausting diarrhœa may also cause extreme prostration, which ends in death. Sometimes the loss of flesh increases to extreme emaciation, and the patient sinks slowly or rapidly. These events may occur when the special symptoms are moderate in degree. Other occasional causes of death have been tuberculosis, cerebral hæmorrhage, rarely hæmorrhage elsewhere, cerebral softening, and pachymeningitis. Sometimes sudden death occurs, the exact cause of which cannot be ascertained.*

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* In another case, characteristic symptoms for six months gradually passed away, and eight years later there was only trifling prominence of the right eyeball, and slight firm enlargement of the left lobe of the thyroid.

PATHOLOGICAL ANATOMY.—In the heart, signs of dilatation and hypertrophy are commonly found after death, but are far less than might be expected from the symptoms present during life. Slight endocarditis is occasionally met with, and graver lesions of the valves are more frequently found than might be anticipated; no doubt because such cases more frequently die. The arteries are found dilated, and may appear to be distinctly hypertrophied (Vogt). The veins also may be enlarged. In the old it is doubtful what significance is to be attached to the state of the arteries.† Aneurismal dilatations have been seen on the arteries of the thyroid, in which all the vessels are often conspicuously enlarged. The proper tissue of the gland is increased in quantity, without being materially altered either in naked-eye or microscopical appearance. Occasionally, however, there is a greater change, either cystic or "colloid" degeneration.

In the orbit the most frequent change is an increase in the quantity of fat, sometimes considerable, but never extreme; in many cases the amount of fat has been perfectly normal. Dilatation of the orbital veins, atheroma of the ophthalmic artery, and fatty degeneration of the ocular muscles, have been met with in rare instances. In one case in which the fatty degeneration of the straight and oblique muscles was very marked, the levatores palpebrarum presented a marked contrast to their normal condition.‡ In this, as in many other cases, no trace of the tissue of Müller's muscle could be discovered. The optic nerve has been found elongated in consequence of the exophthalmos.

The cervical sympathetic has been carefully examined in many cases by competent observers, and has been, as a rule, free from any indication of disease.§ In other cases slight changes have been found; the most frequent has been an increase of connective tissue in and about the lower cervical ganglion, and with this change the ganglion has been described as enlarged, or reduced below the average size, as normal in consistence, or distinctly indurated. An atrophy of the nerve-cells has been described in a few cases. The cardiac ganglia have been found normal when they have been examined. In one case the splanchnic nerves were described as degenerated (Siemerling).

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* Two instances of this are recorded by Hale White, 'Brit. Med. Journal, July, 24th, 1886.

† As, for instance, a case recorded by Wähner, in which thickening of the arteries of the spinal cord was found in a woman of sixty-two.

‡ Silcock, 'Trans. Oph. Soc.,' 1886, p. 104.

§ *E.g.* in thirteen cases reported by Lewin (loc. cit.) it was normal in all.

No significant changes have been discovered in the central nervous system. The medulla oblongata has generally been quite normal; in some cases there have been signs of congestion, and even recent hæmorrhages in or near the sixth, the vagus, and hypoglossal nuclei, and in the restiform bodies.* With the exception of poliomyelitis observed by Drummond, the spinal cord has been found normal, observations not having been made in the cases in which there were indications of its disease. This is also true of the cases with paralysis of the cranial nerves. The other organs of the body are also as a rule normal. Disease of the kidneys has been found in some cases, but in most of these there had been organic heart disease. The thymus and other closed glands undergo no change.†

Pathology.—Two questions are involved in the pathology of exophthalmic goitre, as in that of most other diseases,—the mechanism of the symptoms, and the nature of the malady,—questions that are in part the same, in part distinct.

The dilatation of the vessels and over-action of the heart have directed attention to the sympathetic system of nerves as capable, by its derangement, of giving rise to the symptoms. The cervical sympathetic contains fibres which, when they are irritated, increase the frequency of the heart's action. But the arterial dilatation must be either passive, due to paralysis of the vaso-constrictor sympathetic fibres, or active, due to persistent over-action of the vaso-dilator fibres (Gauthier). For this we must assume a partial affection of the sympathetic, or on the other hypothesis, an opposite disturbance of its different elements. Either seems incompatible with the assumption of any general morbid process in the nerve, such, for instance, as an irritation or inflammation beginning outside the nerve-elements. This consideration has led most recent pathologists to look beyond the sympathetic, to the central nervous system, for the seat of the primary morbid process that deranges the vessels and the heart. This view is supported by the fact that the association of arterial dilatation and frequency of the heart's action is in harmony with the physiological law, discovered by Marey, that there is an inverse ratio between frequency of pulse and general blood-pressure, so that lowered arterial tone, and a frequently acting heart coincide, in consequence of their relation to a central mechanism. These considerations certainly suggest that these two symptoms depend on the derangement of such a mechanism, rather than on a random process in any system of nerves. On the whole, the results of pathological anatomy favour this opinion. The frequency with which no morbid appearance has been found in the sympathetic, and the equivocal character of the changes that have been described in a few cases, seem to prove that these, when present, are of secondary origin, or else are accidentally associated. The point chiefly in recent dispute has been whether a general state of weakness of the nervous system does not underlie the special disturbance, on account of the frequency with which general nerve symptoms are met with.

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* Cheadle; Hale White, 'Brit. Med. Journ.,' 1889; Siemerling, 'Berlin Ges. f. Psych.,' &c., January, 1892. Mendel (ibid.) found a simple diminution in size of one restiform body and the opposite "solitary bundle," probably without significance, unless it entailed a disposition to functional derangement.

† See Hale White, loc. cit.

Leaving, for the present, these general questions, we may consider the probable origin of the special symptoms. The dilation of the heart may reasonably be ascribed to its quick action. The systole is too short to be complete; residual blood gradually entails over-distension and dilatation.* A systolic murmur, such as is common in this disease, is frequently heard in simple dilatation, apart from any mitral regurgitation, which may, however, result if extreme dilatation stretches the auriculo-ventricular orifice beyond the capacity of the valves, and may cease when the dilatation becomes less.

The enlargement of the thyroid was ascribed by Graves to the dilatation of its vessels, and its variation in size with the action of the heart seems to confirm the opinion. It is, moreover, certain that some enlargement of the thyroid will result from vascular distension, such, for instance, as follows ligature of the thyroid veins (Boddaert). According to this view the increase in the proper tissue of the organ, may possibly be a result of the increased blood-supply. Yet two considerations suggest that the relation of this symptom to the primary derangement, whatever this is, may involve more than mere vascular dilatation. (1) The thyroid may be scarcely enlarged when the cardiac and vascular symptoms are very marked; conversely, although rarely, the thyroid may enlarge considerably when the heart is but little excited; and lastly, its enlargement has been known to precede the disturbance of the heart. (2) The mental weakness, &c., that sometimes results from extirpation of the thyroid, seem to show that the organ has a mysterious influence on the central nervous system, and an opposite influence is at least conceivable, if equally mysterious. We must beware of the assumption that the process we can trace is the only one in operation.

The prominence of the eyeballs is difficult to explain, and is probably due to the co-operation of several mechanisms. (1) The increase in the orbital fat, when present, may have a trifling influence. (2) The distension of the vessels of the orbit is probably a more powerful factor; its existence is suggested by the pulsation of the retinal arteries, and its influence, by the variation in the prominence with the over-action of the heart. (3) The unstriated muscular fibres of Müller, which are innervated by the sympathetic, and run from the eyelid to the membranous lining of the orbit, are generally believed to be capable, by their contraction, of causing prominence of the eyeball. The eye recedes a little when the sympathetic is divided, and becomes slightly prominent when it is stimulated. Although it has been questioned whether in man these fibres are sufficiently developed to exert an appreciable effect, it is possible that they may become hypertrophied under permanent excitation. Our present knowledge permits us to invoke no other than these three mechanisms, but it is not easy to conceive how even their combined influence can produce the extreme prominence sometimes seen.

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* See "Dilatation of the Heart," Reynolds' 'System of Medicine,' vol. iv, p. 738.

The loss of descent of the upper eyelid was ascribed by von Graefe to spasm of some of the unstriated fibres of Müller, which pass from the tendon of the levator to the posterior edge of the lid, paralysis of which is supposed to cause the slight ptosis that follows division of the sympathetic. It must be due either to this cause or to a deficiency in the relaxation of the levator which normally occurs as soon as the commencing movement of the globe places a tension on the muscle.* The fact that it does not interfere with closure of the eyes —in which movement the orbicularis at once overcomes the spasm— is more consistent with spasm of the levator from a central mechanism, and it is difficult to refer the retraction of the lid to any influence except that of the levator itself.†

It is a curious fact that cocain applied to the conjunctiva causes a slight retraction of the lids and a defective descent similar to that seen in exophthalmic goitre.‡ It is assumed that the cocain acts upon the sympathetic nerve-endings, since Jessop has found that the effect cannot be obtained a few days after division of the sympathetic in the neck. This does not, however, throw any light on the muscular mechanism by which the symptoms are produced, but it supports the view that they are due to over-action of the sympathetic nerves.

Thus the symptoms seem to be due, partly to the direct effect of deranged action of the sympathetic, and partly to the indirect effect of this derangement on the vascular system.§ It is probable that the cause of the derangement is to be sought beyond the sympathetic in the central nervous system. All the sympathetic functions are under central control, and the character of the symptoms, as we have seen, is in harmony with the physiological relation between the degree of blood-pressure and the frequency of the heart's action. The chief controlling centres, and especially those that determine this relation, are in the region of the medulla oblongata, and it is to this part that we are thus directed in our search for the seat of the disease. The negative results of pathological investigation, and the character of the disturbance itself, make it probable that the morbid state is one of function, and of that finer nutrition that baffles, and will baffle, our means of investigation. Whatever its nature, it is evidently a condition that can pass away even after the lapse of years.

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* This is apparently an example of the relaxation or tension by which the muscles are kept adapted to passive movement. See "The Movements of the Eyelids," 'Med.-Chir. Trans.,' 1879, and 'Lancet,' May, 1890. An explanation that has been suggested, that the phenomenon is due to weakness of the orbicularis, is untenable, because the symptom occurs in the movement in which the orbicularis takes no part, and is absent when the orbicularis contracts, as to close the eyes for sleep.

† See note on the effects of emotion, p. 888.

‡ This fact was pointed out by Koller in his original account of the action of cocain, and the phenomena have been carefully studied by Jessop, 'Trans. Oph. Soc.,' vol. v, p. 240, and vi, 123.

§ A difficulty has been felt by some in conceiving a continuous over-action of the sympathetic, but this difficulty can hardly persist after a consideration of the vascular tone.

Attempts have been made to discover whether experiment can supply the evidence that pathological anatomy fails to afford. Filehne, in particular, has tried to demonstrate the dependence of the disease on the medulla oblongata. He divided the anterior fourth of the restiform bodies, and succeeded in producing exophthalmos, even after division of the sympathetic. In a few experiments he caused also some enlargement of the thyroid, and in one case all three symptoms of exophthalmic goitre were produced. Filehne's experiments have been repeated by others with very similar results.[*]

These results certainly confirm the view which refers the disease to the medulla oblongata. The hæmorrhages found in some cases are doubtless merely indications of functional (and, therefore, of vascular) disturbance. It is possible that some of the cardiac disturbance is produced through the agency, not of the sympathetic, but the vagus. It is important to recognise that the results of experiment derive their chief weight from the indications of physiology, which point to the medulla as the part to which the symptoms must be directly ascribed.

The negative evidence of pathological anatomy, and the positive evidence afforded by the character of the various symptoms, therefore, alike suggest that the malady is a neurosis, a disease of the nerve-elements themselves, having its character determined by their functional relations, and its origin in conditions at present beyond our range of scrutiny. We can thus understand the multiform derangements of the same class with which its symptoms may be associated. But we are still in the dark regarding the way it is produced. Emotion may unquestionably excite it; that is, it may be set up by derangement of the cortex, a fact in harmony with the influence of emotion in exciting the heart, dilating the vessels, and even causing a "staring" appearance of the eyes.[†]

[*] Doudourffis, 'Deut. med. Wochenschr.,' 1887, and Bienfait, 'Bull. de l'Acad. Roy. de Méd. de Belge,' 1890.

[†] The widely open eye of an alarmed animal, on the alert to perceive danger, must be due to the levator, and may be contrasted with the relaxation of this muscle and drooping lid of the sleep that mental tranquillity permits. The excited

The effects of excision of the thyroid—peculiar nerve depression and ultimately general myxœdema* (the "cachexia strumipriva" of Reverdin)—and the coexistence of atrophy of the thyroid with ordinary myxœdema, have seemed to many inseparable from the pathology of exophthalmic goitre. Möbius especially has advocated the theory that a toxic blood-state is produced by the enlarged thyroid, with the excess of mucinoid material in its follicles, and has urged, among other points, that the goitrous idiocy of cretinism must be ascribed to an endemic poison. But even the last opinion rests on no proof, while the great discrepancy between the state of the thyroid and other elements of the disease seems to exclude it from any direct or indirect influence on the symptoms. This is true even of the pyrexia, which has been referred to some toxic material produced by the thyroid (Betoye), but may be due to the direct influence of the disturbed medulla. The occasional beneficial effect of the excision of the thyroid has been adduced in support of the theory of a toxæmic state, due to the thyroid, but the effect is open to too many possible explanations to have appreciable weight. The profound general influence of excision may include the removal of the special disturbance, apart from that of a toxic source. Moreover, if the thyroid can enlarge in consequence of disturbed innervation from the medulla, its removal (as already suggested) may influence the functions of the latter, also through the nervous system. Less unlikely, but still purely hypothetical, is the idea that a primary blood-state disorders the nervous system, put forward by Dr. J. Begbie ('Dublin Hosp. Gaz.,' 1855). It is necessary only to mention the theory of Liebermeister that the thyroid, by compressing the carotids, lessens the amount of blood passing through them—a mechanism which, if possible, would tend to prevent the exophthalmos.

The chief complications, not already mentioned, admit of an explanation in harmony with that of the leading symptoms. The origin of the tremor (which can scarcely be regarded as a complication) is, however, still obscure. It must be due to deranged action of some of the motor cells, but whether those of the spinal cord or the motor cortex of the brain are first disturbed is unknown. It is a constant effect of sudden alarm, but, on the other hand, the occasional disease of the nerve-cells of the cord may indicate a more constant tendency to derangement of their nutrition and function.

The mental disturbance is commonly ascribed to dilatation of the intra-cranial vessels, but it has not been proved that this condition actually exists, or is capable of causing the condition, while other influences cannot be excluded. The general state of nerve weakness must be remembered, and also the possible action on the cortex of

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action of the heart is chiefly conspicuous when motor action, to escape the danger, is impossible, and gives place to tremor. An alarmed horse on a ferry-boat illustrates these conditions in a striking manner.

* See Horsley, 'Lancet,' 1886.

the cause, whatever it may be, which disposes to or excites the disease.

The abnormal vascular state of the skin is distinctly within the range of disturbance of the sympathetic, and the occasional intestinal flux has suggested a like derangement of the intestinal sympathetic nerves. The latter symptom, curiously enough, gives us a clue to the pigmentation of the skin, which has so often attracted notice. The general similarity of this to Addison's disease prevents any weight being placed on the common absence, or occasional presence of pigmentation of the mucous membrane of the mouth. In Addison's disease, the pigmentation certainly depends on disease of the abdominal sympathetic, and on the supra-renal bodies only by reason of their connection with the nerves. In a case of typical Addisonian pigmentation, I found these bodies normal, but the sympathetic plexuses of the abdomen involved in a mass of cancer. The effect commonly due to organic disease may, apparently, be sometimes produced by transient derangement. I have seen a case in which such derangement was suggested by other symptoms, and pigmentation, quite like that of Addison's disease, passed away entirely. Hence an affection of the abdominal sympathetic, similar to the constant affection of the cervical sympathetic, would explain the pigmentation, and the occasional watery diarrhœa seems also to show that the abdominal nerves are sometimes involved.*

DIAGNOSIS.—When all the symptoms of the malady are present, and are considerable in degree, the disease is one that is easy to recognise. It is difficult to detect, and apt to be overlooked, only when it is in an early stage, and partially developed; yet it is in these cases that the diagnosis is of the greatest importance, since it is in these that treatment has most prospect of affording permanent relief. In such cases the chief symptoms are cardiac, and the malady may be mistaken for mere nervous excitement, or for organic heart disease. The strong pulsation of the arteries, and the persistent excess and frequency of the heart's action, are the most important diagnostic indications. A pulse-rate of 120, maintained even when the patient is tranquil, should always arouse a suspicion of the disease, and the thyroid and eyes should be carefully examined.

Ordinary goitre is distinguished by the absence of the cardiac and ocular symptoms, by the degree of enlargement of the thyroid, and by its relation to endemic influences. But there is a class of cases with cardiac symptoms which most authorities consider ought to be kept distinct from the true exophthalmic form. A large goitre of endemic origin becomes associated, after a time, with over-action of the heart, and exophthalmos, in consequence of the pressure of the tumour on the sympathetic, and perhaps also on the vagus. The exophthalmos is usually one-sided, corresponding to the chief enlargement of the thyroid, and is accompanied with spasmodic mydriasis, from irritation of the sympathetic fibres to the iris.

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* Lesions of the solar plexus are said to cause persistent watery diarrhœa (Cristiani, 'Riforma Med.,' September 17th, 1891).

PROGNOSIS.—In few diseases of equal severity and corresponding apparent gravity is the prognosis so uncertain or do so few cases end fatally under observation. Yet it must be confessed that when the eyes are distinctly prominent, the thyroid enlarged, the heart dilated, and its habitual frequency much increased, it is rare to observe more than a moderate degree of improvement as the result of the most careful treatment. In many cases, improvement is followed by relapse, and in some no amelioration can be obtained. On the other hand, some cases present a degree of improvement that amounts almost to restoration of health, and perfect recovery is occasionally observed. The prospect is best in the slighter and earlier cases, in which the heart disturbance is recent, and the enlargement of the thyroid is slight. The prognosis is, on the whole, less favorable the more severe the symptoms, also when there is conspicuous failure of general nutrition, or organic heart disease. It is better in women than in men; nevertheless cases of recovery in men are sometimes met with.* It is better if the disease shows a remittent tendency, and I am inclined to think that, if the disease is in an early stage, it is better when there is a family tendency to the affection than when there is none. A distinct and removable exciting cause certainly also improves the prognosis. If no improvement is obtained, an opinion is often sought regarding the probable duration of life. This is, however, impossible to forecast. The sufferers may present merely variations in the degree of the symptoms, and even then an unexpected improvement may occur for month after month and year after year. One patient, when I saw her, was in a condition in which it seemed scarcely possible that she could live three months, and yet three years afterwards she was not only alive, but so much better that she had become a wife and mother. Several other instances of similar improvement have come under my notice. On the other hand, a case of only slight degree may be treated sedulously for months with only the result that the symptoms do not increase. The average prognosis is certainly worse among hospital patients than in those of better position, in whom suitable conditions of life can be secured and maintained for a long time. But in this, as in so many diseases, the observed tendency is the only trustworthy guide to prognosis, and it is never right to exclude the hope of ultimate improvement and even of possible recovery.

TREATMENT.—The first and most important element in treatment is to secure tranquillity of mind and rest of body. Frequently as the heart, in this disease, beats during rest, it is quickened still more by mental emotion and physical exertion, and every avoidable contraction should be prevented. In a severe case it is well to keep the patient in the recumbent posture for a time—in bed if the onset has been recent and acute, or if the symptoms are severe. Absolute rest may quickly bring the pulse down from 120 to 90, or even 80. Rest often enables drugs to do good which before were powerless. In any case, several hours of each day should be spent on the sofa, and only the gentlest exercise should be permitted. If perfect rest is desirable, the lack of exercise may, in some degree, be compensated by gentle daily massage. Complete change of air has often been followed by marked improvement, but as a rule mountain air is not desirable. Most patients do best at the seaside, if the air of the place is not too stimulating. A sea voyage is a somewhat hazardous measure. In each case the effect of change must be carefully watched, since it is not alway possible to foretell its influence. There is no trustworthy evidence that any particular locality is specially beneficial.

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* See 'Trans, Oph. Soc.,' 1886, p. 26.

Medicinal treatment is uncertain in its results. Drugs that have clearly done good in some instances have been quite ineffectual in other cases. This statement applies to iron, quinine, arsenic, strychnia, and other tonics. In spite of anæmia, iron fails in most cases to improve the condition of the patient, and seems sometimes even to do harm. Von Graefe, whose experience of the disease was very large, came to the conclusion that iron is useless in the height of the malady, but that it does good when a certain amount of improvement has occurred, and the pulse has fallen below 100. Iodine and iodides have been thought by many to be positively harmful; nevertheless, cases are occasionally reported in which benefit has followed their internal administration or external use. Bromides seldom influence the disease itself, but are often useful for the purpose of lessening irritability and promoting sleep. Antipyrine has been strongly recommended by Gauthier, but sulphonal has been found by many the most valuable hypnotic (gr. 15—25); it is said to lessen the over-action of the heart, and to some extent the other symptoms, if given twice a day in 10 gr. doses. But it should not be thus administered for long.

Drugs that slow the pulse would seem to be specially indicated, but the experience of different observers is as much at variance on this point as on the action of tonics. It is certain that digitalis fails, in most cases, to produce any permanent effect, and yet cases are occasionally published in which digitalis and iron together have seemed to cure the patient. Trousseau thought digitalis useful when given in full doses, so as to reduce the pulse to sixty or seventy beats per minute. Strophanthus has been said often to do good, and to be free from risk of harm.* Veratrum viride does not seem to possess any special advantage. Aconite has been recommended in doses of five minims of the tincture, increased gradually to twenty.

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* Fergusson, 'New York Med. Rec.,' 1890; Möbius, loc. cit.

Belladonna, however, is the drug that has seemed most often distinctly useful. It should be given in gradually increasing doses, until the maximum quantity is reached that the patient can bear. Either the tincture or the extract may be given, or atropine may be injected. Pronounced symptoms may disappear entirely under its use, and still more frequently marked improvement results. Both belladonna and digitalis, it may be noted, have an influence on the small arteries, increasing their tone. Ergot has rarely failed to disappoint. Prolonged hydropathic treatment has been strongly praised, chiefly by continental physicians.

The application of voltaism to the cervical sympathetic has been recommended, and often employed. It is not certain to what extent the sympathetic is influenced, since we cannot by such application to a healthy individual, obtain any dilatation of the pupil. The application is followed in many cases by a distinct fall in the frequency of the pulse (amounting to ten or even twenty beats per minute), and sometimes by a slight diminution in the size of the thyroid. I have employed the treatment in many cases and have known the pulse to fall twenty or thirty beats. The effect, unfortunately, is usually transient, and although repeated applications may sometimes cause a slight degree of permanent improvement, this is, as a rule, trifling. When all the symptoms have disappeared, other measures were generally employed at the same time, and the treatment continued for a long period. Both electrodes may be placed along the anterior edge of the sterno-mastoid, one above and one below, or one electrode may be placed in either of these positions and the other on the lower cervical spines or beneath the occiput. A weak current only should be employed, such as causes a moderate amount of tingling in the skin. Faradism from the neck to the face, thyroid, and cardiac region, has been advocated by Vigouroux and Charcot.

Surgical Treatment.—Excision of the thyroid is scarcely practicable when the disease is fully developed. The dilatation of the blood-vessels renders the operation very formidable, while it has other special risks, immediate and remote. In a few cases, chiefly early, it is said to have been successful in arresting or curing the disease, and in some other cases in which an operation was justified by threatened suffocation, but in most the enlargement of the thyroid presented some peculiarity and differed from the ordinary extremely vascular form In this disease, as in ordinary goitre, excision of the isthmus causes atrophy of the whole gland, but the operation has not been followed by permanent results to the other symptoms of the exophthalmic form. Excision has failed to influence the symptoms in some recorded cases. In one patient on whom the operation was performed, the resulting atrophy of the gland did not materially influence the exophthalmus, and there was also considerable valvular disease of the heart, which caused death four months later.* The same may be said of setons, cauterisation, and injections of ergotin. Handfield Jones has seen injections of iron into the thyroid give rise to alarming symptoms. On the whole, the results of surgical treatment are not at present encouraging, but it is possible that the future may disclose the means of making it more effectual. Especially it deserves note that ligature of the arteries of the thyroid is said to have the same effect as extirpation, and to augment, to three times the normal, both the absorption of oxygen and the formation of carbonic acid.*

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* Bristowe, 'Trans. Oph. Soc.,' 1886. The large proportion of published cases

Among special symptoms that may call for treatment, the paroxysms of palpitation and dyspnœa are best relieved by "derivatives," as hip-baths and mustard plasters to the feet, with cold to the thyroid (if its enlargement is sufficient to be a cause of dyspnœa) and by full doses of digitalis. Ether, chloroform, and morphia also sometimes give relief in this condition. Trousseau found a copious venesection effectual in relieving such an attack when other measures failed. Any inflammation of the eyeball must be promptly treated, to obviate, if possible, the danger to sight.

PARALYSIS AFTER ACUTE DISEASES.

Many acute diseases, especially the acute specific diseases, are occasionally accompanied or followed by symptoms of impairment of function of the nervous system. Of these symptoms, motor palsy is the most frequent. One of these diseases, diphtheria, is followed by palsy of a special character, which will be separately described. In the other maladies, the nervous symptoms vary much in their character, and evidently also in their mechanism. They are on the whole rare, but less so after some diseases than after others.

In the course of any acute disease, sudden symptoms may come on, such as indicate the occurrence of a vascular lesion in the brain. Usually there is sudden hemiplegia, which may be accompanied by loss of speech, and may persist or may pass away according to the position of the lesion. In some cases, especially in children, convulsions attend the onset, but chiefly when the lesion is cortical; possibly the greater frequency of convulsions in children is because the central lesion is in them more frequently at the surface. The morbid process varies in different cases. In some there is embolism, consequent on endocarditis, which may complicate any one of these diseases, but is most frequent in scarlet fever. In other cases, the lesion is probably thrombosis in an artery or in a surface vein, and the latter is especially probable in the case of children. In other very rare

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in which benefit resulted suggests that a still larger number, without benefit, have been unpublished.

* Tarchanoff, 'Schweiz. Corr.-Bl.,' 1889.

cases, again, there is hæmorrhage. All such secondary cerebral lesions are most common in children.

The other symptoms met with present no general correspondence in character; they may be cerebral or spinal, or such as indicate an affection of the peripheral nerves. In other cases the symptoms are those of functional disturbance, sometimes prolonged. The affections of the nervous system which occur during the acute disease, besides the vascular lesions already mentioned, are generally inflammatory, and seem due to the influence of the virus of the primary disease, which often causes inflammation in other organs. Those that follow the disease vary not only in their character, but also in the date at which they occur. Many facts make it probable that they are due to some product of the organisms which may be safely assumed to constitute the virus of most of the primary diseases. This product or "toxine" has certainly sometimes a powerful action on the nervous system (see "Tetanus, Pathology"), and it is probably the agent by which most sequelæ are produced that are not the result of the simple debility caused by the acute malady.

Typhoid Fever.—Apart from the sudden hemiplegia already mentioned, which is very rare in typhoid fever, the most common nerve-symptoms indicate a special influence of the poison on the brain. The common deafness is apparently produced in this manner by an interference with the functions of the auditory nerve. Loss of speech is not uncommon. It occurs chiefly towards the close of the disease and during convalescence; it may come on as the deafness is passing off. There is generally complete speechlessness, and, although it is called aphasia, there is no disorder of speech, or partial loss, such as occurs in cases of organic disease of the brain. The patient can understand perfectly what is said, and may be able to express assent and dissent by signs, or even to write. In one case under my observation the condition developed gradually by a disuse of the lips, which were dry and cracked; the loss of the labial articulation rendered the words almost unintelligible, and they gradually ceased to be uttered. The condition may last for some weeks. In one recorded case it was associated with general choreoid movements.* Not unconnected, probably, with such loss of speech, is the mental weakness that so often attends convalescence from typhoid fever, and may amount to actual imbecility. Impairment of memory is common. Occasionally there is some more pronounced mental derangement, generally in the form of chronic delusional insanity. These cerebral symptoms always pass away in time, but in adults the memory may never be so good as before the fever.

Paraplegic weakness is not uncommon, and this also occurs when the disease is on the decline or during convalescence, or at least is then recognised. In some cases, the weakness due to the fever seems to persist in the legs, and, as in one case under my observation, when the patient is seen some months after, there is distinct defect of power, with excessive knee-jerk and foot-clonus.[*] Money has indeed found that there is generally an excess of myotatic irritability during the disease.[†] In other cases actual paraplegia comes on more rapidly, although not suddenly, and may increase to a considerable degree of weakness, although there is rarely absolute loss of power. The onset may be attended with spinal tenderness, and with hyperæsthesia or various subjective sensations in the legs, and these may be followed by defect of sensation. It is probable that the symptoms are due to disseminated myelitis of slight intensity. They usually pass away in the course of a few weeks. In very rare cases paralytic symptoms are of greater severity. The weakness, commencing in the legs, may invade the arms, and when such extension occurs rapidly the case becomes one of the so-called "acute ascending paralysis," and death has been known to occur in the course of a few days.

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* De la Harpe, 'Rev. méd. de la Suisse Rom.,' 1883, No. 6.

Limited atrophic paralysis occurs in some cases, especially during convalescence. It is sometimes very severe, and may leave permanent disability. In acute cases the lesion is probably always spinal, anterior polio-myelitis. There is loss of power, variable in extent, with rapid wasting and loss of faradic irritability in the muscles most affected. Although a considerable amount of recovery always occurs, there may be permanent atrophy of certain muscles. In one case of such acute atrophic paralysis, affecting only the extensors of the wrists and fingers (as in lead-poisoning, &c.), with rapid loss of all irritability, Shore found acute inflammation of the anterior cornua from the third to the eighth cervical nerves. The onset was three weeks after the typhoid, and the patient died from pneumonia two weeks later.[‡] Polio-myelitis is more frequently secondary to typhoid fever than to any other acute specific disease. When the onset is subacute it is probable that in many cases such symptoms are due, not to polio-myelitis, but to multiple neuritis. This was probably present in a case described by Alexander, in which typhoid fever, in a patient aged twenty, was followed by wasting of muscles with loss of electrical irritability and of the knee-jerk, and paralysis of one vocal cord. The development of the palsy was accompanied by severe pains in the legs.[§] Recovery was complete. Such symptoms have probably been due to neuritis in cases in which they involved all the extremities, since this sequel has also been proved. The distinction of the central or peripheral nature of many cases is very difficult, and depends chiefly on the presence of pains in the hands and of nerve tenderness. In some cases of local atrophy, as of one arm, pains and tenderness have made an isolated neuritis probable, which may sometimes be excited by even slight pressure.

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[*] It is not uncommon for adults after typhoid never to regain the good walking powers they had before the illness, although there is nothing that can be called paralysis.

[†] 'Lancet,' 1885.

[‡] 'St. Bartholomew's Hosp. Rep.,' xxiii.

[§] Alexander, 'Deut. med. Wochenschr.,' 1886, No. 31.

In very rare instances paraplegic or general weakness after typhoid fever has been associated with paralysis of the soft palate, but even these cases do not resemble closely diphtheritic paralysis. There is never the loss of accommodation, or the slow progress of palsy from one part to another.*

Paralysis of cranial nerves has been seen in a few instances, and in two cases, paralysis of the abductors of the vocal cords occurred in so complete a degree as to necessitate tracheotomy (Rehn, Villemin). Optic neuritis is a very rare sequel of typhoid fever. Localised neuralgic pains in the course of the spinal nerves are occasionally met with during convalescence. They have been known to be accompanied by spots of anæsthesia, and are then doubtless due to neuritis. Landouzy records a case in which a spot of such anæsthesia in the groin was permanent.

The muscular tremor, which is occasionally so conspicuous during the course of typhoid, may continue during convalescence, and it has been known to persist and to be followed by the symptoms of disseminated sclerosis (Chauffard, Ebstein, and Westphal). The tremor never goes on to paralysis agitans. In girls, hysterical symptoms are not rare during the course of the disease and during convalescence; contractures, anæsthesia, and even convulsions have been met with.

Typhus Fever.—After typhus fever, symptoms have been met with very similar to those that may follow typhoid. Paraplegic weakness is, however, less common; paralysis of cranial nerves occurs only in consequence of an attack of meningitis complicating the fever; while paralysis of the palate has not been met with. Bernhardt has recorded a case of neuritis of the musculo-spiral nerve, which may have been set up, as paralysis in this nerve so often is, by pressure upon it during the period of prostration and inertia. Rarely symptoms of multiple neuritis have followed typhus. Optic neuritis has also been observed, although even more rarely than after typhoid.

Erysipelas is seldom followed by paralysis. In one remarkable case† paralysis of the soft palate and of the posterior crico-arytænoids (abductors of the vocal cords) followed facial erysipelas and one-sided pneumonia. The paralysis of the larynx necessitated tracheotomy, and appears to have been permanent.

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* True diphtheria sometimes complicates typhoid fever, and may be followed by characteristic paralysis, as in a case related by Dr. Murchison. In the cases in which membrane in the throat has been regarded as an effect of the typhoid, it is possible that the virus of diphtheria may have been also present. This point deserves further consideration in the cases that are followed by palatine palsy, especially when there is general muscular weakness. (See Ross, 'Congress American Phys.,' 1888.)

† Feith, 'Berlin. klin. Wochenschrift,' 1874, No. 49.

VARIOLA.—Spinal symptoms are more common in connection with smallpox than with any other of the eruptive fevers. The fact was associated by Trousseau with the severe pain in the back which occurs at the onset, on the ground that this pain may be accompanied by weakness, formication, and tingling in the legs, plantar pain, and retention of urine, and that these symptoms pass away with the pain in the back on the appearance of the eruption. More pronounced spinal symptoms may come on during the height of the disease, or especially during convalescence. There is paraplegic weakness, with or without affection of sensation, and varying much in degree. Recovery in severe cases may be incomplete. The symptoms probably depend upon myelitis, which, disseminated through the cord, was actually found by Westphal in one case; the grey and white substance was occupied by scattered spots of softening.* Acute atrophic paralysis has also been observed; it is sometimes due to polio-myelitis (as Roger was able in one case to demonstrate), but probably in many cases, and perhaps in all those in which the onset is subacute, it is the result of neuritis. In a case of atrophy in one arm, Joffroy could find no change in the cord, but only an acute degeneration of the nerves. In many other cases, the characteristic symptoms of multiple neuritis, affecting all the limbs, and sometimes other parts, have come on at an interval after the variola (see vol. i, p. 114). Ataxy of both arms and legs, persisting for a long time after the primary disease, but ultimately passing away, has probably been due to the same cause. Acute ascending paralysis has been observed during convalescence, fatal in a few days, and, as in other cases, without recognisable lesion.† Occasionally paralytic symptoms extend to the cerebral as well as the spinal functions. The ocular muscles have been paralysed, perhaps from neuritis. Smallpox has also been followed by symptoms of insular sclerosis.‡

MEASLES.—Spinal symptoms may occur during and after measles, very similar in characters to those which follow smallpox. Those of acute polio-myelitis are the most frequent, but acute ascending paralysis has been met with (Bergeron and Liégeard). A remarkable case of disseminated myelitis has been recorded by Barlow.§ Hemiplegia, probably from thrombosis in a cerebral vessel, has also been met with. Optic neuritis has only been observed as a result of coincident meningitis.

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* Foci of softening have also been described by Damaschino, and by Joffroy and Hayem.

† Bernhardt, 'Berl. klin. Wochensch.,' 1871, No. 47; Gros and Beauvais, 'L'Un. méd.,' 1884, No. 131.

‡ Long, Charcot.

§ 'Med.-Chir. Trans.,' 1885.

Scarlet Fever.—Spinal symptoms are rare after scarlet fever; sudden cerebral hemiplegia is not infrequent, and is probably due to embolism from scarlatinal endocarditis, or to spontaneous thrombosis. It may occur during or apart from kidney sequelæ. Convulsions, which may persist as epilepsy, are also more frequent after scarlet fever than after any other acute disease (see p. 733). Neuro-retinitis has been many times observed to follow scarlet fever, without any renal complication.*

Mumps.—Palsy of accommodation has been described, but was met with only in one case in an extensive epidemic.† Joffroy observed paralysis of the limbs in one instance; there was loss of electrical irritability and of reflex action, and the muscles were tender.‡ It is possible, however, that in each of these cases there may have been undiscovered diphtheria; in the second case, indeed, slight angina was observed.

Malarial Fever.—During the course of severe malarial fever singular paralytic symptoms have been met with, especially hemiplegia and aphasia, which are said to pass away under the influence of quinine, and are therefore ascribed to an effect of the malarial poison on the nerve-centres (Grasset). Graver and more lasting symptoms of the same class have been met with in cachectic subjects, and have been ascribed to obstruction of cerebral vessels by pigmentary matter. The most frequent paralysis due to malaria is that of the feet, less commonly also of the hands, which is characteristic of multiple neuritis. This form has been described in vol. i.

Dysentery, when severe, has been followed by paraplegia, commencing with tingling in the skin, and pains along the course of the nerves, succeeded by motor and sensory paralysis, sometimes with muscular wasting. The arms may also be involved. These symptoms sometimes occur during the course of the disease. They are probably due to myelitis or to peripheral neuritis. In one case, extensive softening was found in the cervical and lumbar enlargements (Delioux).

Diarrhœa.—Simple diarrhœa, whether spontaneous or the result of undue purgation, has been followed by spinal symptoms, usually transient, sometimes more severe. In women, hysterical symptoms also result from the peculiarly depressing effect of diarrhœa on the nervous system. Tetany is an occasional sequel, especially in children, in whom exhausting diarrhœa sometimes causes also cerebral thrombosis, or the symptoms of cerebral anæmia termed "hydrocephaloid" (see p. 370).

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* In scarlet fever there is sometimes false membrane in the throat like that of diphtheria, and the patients are often said to have had both diseases. My colleague Dr. Barlow, whose experience of these cases is very large, informs me that he has never known diphtheritic paralysis to follow this complication, and hence it is very doubtful whether the membrane is an indication of true diphtheria. It is possible, however, that there are two forms of this complication, one truly diphtheritic (see Heubner, 'Jahrb. f. Kinderheilk.,' Bd. xiv; and Demme, 'Bericht aus d. Jennerspital,' Berne, 1878 and 1879).

† Boas, 'Kl. Monatsbl. f. Augenh.,' 1886, p. 273.

‡ Joffroy, 'Prog. méd.,' 1886, No. 47.

Acute Rheumatism.—No palsy is common in connection with acute rheumatism, except the embolic hemiplegia that is the result of endocarditis. Myelitis, especially polio-myelitis, has been thought to be a frequent sequel, but the cases on which this opinion has been based have been, for the most part, those in which pains in the limbs have attended the onset of the spinal disease, and have given rise to an erroneous diagnosis of acute rheumatism. It is possible, however, that rheumatism and myelitis may both result from the same exposure to cold, which may certainly cause arthritis and polyneuritis as coincident effects (see vol. i). A patient aged thirty had subacute articular rheumatism of both ankle-joints, with considerable pain, and this was followed by incomplete palsy of all limbs, disturbance of sensibility, partial atrophy of the muscles with the degenerative reaction, loss of the knee-jerk, tremors, and involuntary twitchings; the facial nerves were also involved, and some optic neuritis supervened.*

INFLUENZA.

There is no acute malady, with the exception of diphtheria, after which disturbance of the nervous system is so frequent as after influenza, and there is certainly no disease that has such varied nervous sequelæ. This effect of influenza, although long known, has never been perceived so distinctly as in the severe outbreak of the last four years, 1890–93. Functional disturbance of the nervous system seems, indeed, to be a direct effect of the virus of the disease, since it forms an almost constant feature of the acute affection. That which exists during the febrile stage may persist in similar form for many days or weeks after the general disturbance is at an end. Symptoms of similar or different nature may develop within a fortnight of the cessation of the acute attack; and, lastly, during this, or afterwards, organic disease may develop in the central organs or peripheral nerves. Some of the most severe sequelæ, moreover, have followed second or third attacks of influenza which were mild, and sometimes even trifling. On the other hand, there has been a disposition to associate with influenza affections of the nervous system which have come on six, nine, or twelve months after the primary disease, without any other connection with this than some impairment of constitutional strength, such as follows every depressing acute illness. Such remote affections cannot be regarded as the specific consequences of influenza. Moreover, many of the direct sequelæ cannot be regarded as quite special, since so often a previous disposition to them can be distinctly traced.

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* Remak, 'Neur. Centralbl.,' 1885, No. 14.

The mental state and physical depression or inertia, seldom absent, rarely cease with the acute attack; this almost universal sequel has become familiar to every one. It is not surprising that, especially in predisposed individuals, definite melancholia should grow out of it, sometimes of the hypochondriacal type, sometimes with definite delusions, and occasionally with suicidal impulses. Delirium occurs, though not frequently, in the acute stage, and there may be subsequently a period of mental derangement with excitement, sometimes with hallucinations; it is very rare for this condition to amount to actual mania. Mental dulness is more frequent, and, in the old, it may amount to positive dementia, which is sometimes actual mindlessness. Very seldom influenza has been followed by advancing symptoms of general paralysis of the insane, chiefly when commencing symptoms had previously existed. With the exception of the last, and of senile derangement, these psychical sequelæ are usually transient, melancholia being the most prolonged.

Of other forms of functional disturbance, hysteria is very common in those who are predisposed to it, and has been met with in men and in children, and in almost every phase. A state of trance, simple or cataleptoid, has occurred immediately after the febrile stage. Convulsions of epileptic character have commenced soon after influenza, or have then recurred after a long period of freedom; and in one case the acute illness had scarcely ceased when the symptoms of idiopathic tetanus developed and caused death in seven days.

Corresponding to the prominence of pain, especially in the head and back, as a symptom of influenza, we meet with various forms of neuralgia as frequent sequelæ. Some of these are at first very severe, attended with local tenderness, and so persistent as to make it probable that there was, in the beginning, a definite neuritis. The intercostal nerves, the nerves of the arm, and the sciatic nerve are not uncommon seats of such symptoms. Apart from such isolated neuritis, many cases have been recorded in which the characteristic indications of multiple neuritis came on, one or two weeks after the primary disease, and continued for several weeks or even months, passing away, however, as a rule, sooner than those of common toxic origin. In such cases there have been, besides symmetrical nerve-pains and tenderness, muscular atrophy with the reaction of degeneration, loss of the knee-jerk, paræsthesia, and sometimes impairment of sensibility; as a rule, however, the sensory fibres have seemed to suffer less than the motor.

Especially in patients who inherit or have acquired gout, or have suffered from rheumatism, severe and very obstinate forms of what may be termed "fibrous rheumatism" are occasionally set up by influenza, commencing soon after the latter has terminated, but continuing for many months, and even, in late life, passing into a malady likely to end only with life. Pain on movement—in some part of the upper limbs, or especially of the trunk, the loins, or one lumbar and inguinal region, and often the hip and thigh—is associated with spontaneous pain and diffuse tenderness. There may be greater pain and tenderness along one or more nerves in the region, suggesting their implication. The pain and tenderness may involve the spine, but not in equal degree with the other parts, and often with similar extensions. Such symptoms continue, but without any further development. In other cases, moreover, such pains in the fibrous tissues become especially severe about the joints, and these may become distinctly inflamed. In some, the hip or knee, in others, the finger-joints thus suffer; in the latter case both hands are involved; there is arthropathic wasting of the muscles, and the malady may assume the aspect of pronounced rheumatoid arthritis.

Grave inflammations occasionally develop in the nerve-centres, usually during the febrile stage of the disease, as a result of special intensity of its virus. They are comparable to the pneumonia that occurs at the same stage, and is often associated with these affections of the nerve-centres. The latter comprise cerebral meningitis, hæmorrhagic cerebritis, myelitis,—chiefly in the cervical region and associated with meningitis, or else disseminated, occurring in scattered foci throughout the cord. These affections often cause death. Although they commence during the acute stage of the disease, and occasionally very soon after its onset, their effects may only be obvious after that stage is or should be over, by a persistence of cerebral disturbance—which may at first have been ascribed to the derangement of function by the blood-state—or by the supervention of such symptoms when the patient seems passing into convalescence, or of other symptoms, such as acute paralysis, which are unlike those that are met with in the acute stage.

These nerve disturbances seldom involve the cranial nerves. Only in multiple neuritis has facial paralysis and conspicuous difficulty of swallowing been observed. Although infrequency of the heart's action, and vomiting without nausea, may, during the febrile stage, show a special action of the poison on the centres of the medulla oblongata, these effects are not usually prolonged. In the eye, however, optic neuritis has been occasionally observed immediately after the acute affection, as after so many other acute diseases, and the influence of the blood-state is also sometimes manifested by retinitis with spots like those of the renal form, both when there is no albuminuria and when there is, and also with or without inflammation of the papilla. Retro-ocular neuritis has also been met with, attended with only very slight visible inflammation.

The treatment of these varied sequelæ of influenza presents no special features; it is the same as that of the same affections when they occur in consequence of other causes.

DIPHTHERITIC PARALYSIS.

Diphtheria is frequently succeeded by paralysis of definite and peculiar character, such as is not met with after other acute diseases, or is met with only in extremely rare instances. The occurrence of paralysis after low forms of sore throat has long been known, and was described by the elder Chomel as observed in an epidemic at Paris in 1748, by Ghisi in Italy in 1749, and by Bard at New York in 1771. When diphtheria came to be distinguished from other forms of angina, it alone was found to be succeeded by this paralysis. Hence it is almost certain that the epidemic sore throats which, in the last century, were followed by paralysis, were also diphtheria. It is, indeed, believed by some that a similar paralysis occurs, in rare cases, after simple or herpetic tonsillitis, but the evidence of this is not conclusive.* It is, on the whole, probable that all sore throats followed by paralysis are diphtheritic. At the same time other maladies are occasionally followed by paralysis, due to a process similar to that which causes this form, but the nature of the primary disease has usually been distinct. They have been already mentioned.

The frequency with which diphtheria is followed by paralysis seems to vary in different epidemics and at different ages; it has been variously stated at from 8 to 66 per cent. It is probably not far from the truth to say that, on an average, one fourth of those who do not die from the primary disease subsequently suffer from paralysis. Its occurrence is not influenced by sex, but, according to the statistics collected by Landouzy,† age distinctly increases the liability to suffer. The sequel may, indeed, occur at any age, from two years upwards, but adults furnish a larger proportion of the cases of paralysis than of diphtheria. Sixty-eight cases of paralysis were distributed as follows (Landouzy):—Under 10, fifteen; 10—20, eighteen; 20—30, thirteen; 30—40, eight; 40—50, eight; over 50, six. From this it would seem that the tendency to paralysis is very slight in early infancy, and that it increases the older the sufferer. I have seen one severe case at the age of sixty.

The previous health of the patient seems to have no influence; paralysis does not occur more frequently in the weak than in the strong.

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* Cases, however, have been met with (see p. 917) that suggest that a paralysis closely analogous to diphtheritic paralysis may occur as a primary affection without any preceding acute disease, and may run a course similar to that which succeeds diphtheria. I have seen one or two cases suggestive of this conclusion.

† 'Des Paralysies dans les maladies aiguës,' Paris, 1880.

Nor does the severity of the attack of diphtheria determine the occurrence of palsy; it is equally likely to follow attacks that are severe and those that are trifling. It occurs after diphtheria of the surface as well as after that of the throat. The celebrated alienist Griesinger died from paralysis succeeding a diphtheria limited to the wound produced by opening a perityphlitic abscess.

Time of Onset.—As a rule the primary disease is over before the paralysis comes on, the most common time being the second or third week after the termination of the throat affection. Sometimes, however, it commences during the course of the diphtheria, from the fifth to the tenth day, and it has been known to commence as early as the second day. These statements apply to definite loss of power.

SYMPTOMS.—The onset of the paralysis is usually marked by no general symptoms, but now and then a slight elevation of temperature, and, in children, restlessness and irritability, may precede it, and they may also accompany its subsequent extension. The first part to be affected is usually the palate, and fluids are found to regurgitate through the nose. Vision for near objects is then interfered with, in consequence of loss of power of accommodation. These two palsies are almost invariable. Subsequently, and less frequently (in about half the cases), the limbs become weak, and often they are the seat of numbness or tingling, and sensation may be definitely impaired. Less frequent are paralyses of the muscles of the trunk, neck, and larynx, disturbed innervation of the heart, weakness of the external ocular muscles, of the bladder and rectum, and loss of sexual power.

The characteristic of diphtheritic paralysis is its gradual onset and progress in each part, and the way in which different and often distant parts are affected, one after the other. The loss of motor power is accompanied, and indeed preceded, by a loss of myotatic irritability, evinced, in the case of the legs, by the loss of the knee-jerk. When the weakness is considerable, the electric irritability of the muscles is usually changed, and there is moderate wasting. The fact that the knee-jerk disappears before the onset of the palsy is part of a wider fact, that the knee-jerk is often lost after diphtheria in cases in which no paralysis occurs. This was discovered by Bernhardt,* and has since been abundantly confirmed. He found the knee-jerk was lost in no less than two thirds of the cases examined (twenty-two in number), in which there was no definite palsy. The loss occurred usually during the second month, sometimes towards the end of the first month, *i. e.* three or four weeks after the onset of the primary disease. It both disappeared and returned first on one side, and in one case the loss was unilateral.† Sometimes, however, the knee-jerk is lost during the acute disease. Even when there is no paralysis, months have elapsed before it could be obtained.

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* 'Virchow's Archiv,' 1885, Bd. xcix, p. 293.

† According to Money, the loss may be preceded by a brief exaggeration ('Treat-

The paralysis of the palate is shown by the voice acquiring a nasal tone, and by the partial regurgitation of liquids through the nose during deglutition. The nasal tone is of course due to the cavity of the nose not being shut off during phonation; and hence also the explosive consonants are imperfectly pronounced: *p*, for instance, becomes *m*, and the patient is unable to distend the cheeks or to blow out a candle. The closure of the anterior nares at once removes this disability. Gargling also is no longer possible. On inspection, the palate is seen to hang more vertically than normal, and when the patient utters the sound "ah" it is not raised as in health. One side is sometimes lower than the other, but it is rare for one side to be paralysed alone. There is usually distinct loss of sensibility in the palate; the patient does not know when it is touched, or the touch occasions no discomfort or reflex action, although, if the back of the pharynx is touched, evidence of sensibility is at once obvious. Very rarely anæsthesia has been observed without motor palsy. When the paralysis has lasted some time, the palatal muscles cannot be stimulated by faradism as readily as in health.

Paralysis of the pharynx is not common, but may occur, and may be so considerable as to render swallowing almost impossible. The pharynx has even been relaxed to such a degree as to form a pouch in which food accumulates. If it occurs early it is a grave sign. In the larynx, paralysis of the upper part (superior laryngeal nerve) is more frequent than that of the vocal cords (inferior laryngeal nerve). In the former case the epiglottis stands erect against the base of the tongue, and does not descend over the opening, during the act of deglutition, in consequence of the weakness of the depressors. The upper part of the larynx is insensitive, although when a foreign body reaches the vocal cords pain is felt. Hence food is apt to get into the larynx and to cause coughing. The voice is hoarse, probably in consequence of paralysis of the crico-thyroid muscle, but the vocal cords move as usual. In other cases there is paralysis in the region of the inferior laryngeal nerve, and phonation may be impossible. The laryngoscope then shows immobility of the cords and sometimes a preponderant weakness of abduction, so that the cords are not separated during inspiration. In one fatal case, at the end of the first week, swallowing was impossible, and there was complete motor and sensory paralysis of the larynx.* One vocal cord is seldom affected alone, but the left was motionless, while only the tensors were feeble in the right, in a case in which the palate was paralysed on the left side and much weakened on the right.†

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ment of Dis. in Children,' p. 515). Bristowe has observed it to persist during paralysis, and afterwards for a time to disappear, no doubt on account of the special local variations in the incidence of the toxic influence.

* Caiger, 'Lancet,' ii, 1889.

† J. B. Ball, 'Lancet,' 1890.

The tongue is paralysed only in rare cases. Paralysis of the face is also rare, but there is occasionally distinct loss of power on one side, and the lips are sometimes weak. Very rarely there is considerable bilateral paralysis, either of all parts of the face, or of the lower portion only.

Special Senses.—Next to the affection of the palate, both in frequency and in time, is loss of power of accommodation of the eye, from paralysis of the ciliary muscle. This is always described by the patient as "failure of sight," but only vision for near objects is impaired. It has been alleged that the "far point" is more distant than under normal circumstances, but this statement is probably erroneous.* The degree to which sight is affected depends on the refraction of the eyeball, *i. e.* on the degree to which the power of accommodation is habitually employed. With normal refraction, near vision is considerably interfered with; the patient, for instance, cannot read, while distant vision remains good. In hypermetropia this effect is still more marked; while if there is myopia vision may suffer little or not at all, because the patient does not need to accommodate. Before inferring the absence of paralysis from the absence of affection of sight, it is therefore necessary to ascertain that the patient is not myopic. Both eyes are always affected, although one may suffer before the other. The failure of accommodation reaches its height in a few days, and usually lasts for two or three weeks. The action of the pupil to light may be sluggish, but is rarely lost; usually there is contraction on an effort at accommodation, even when no accommodation is possible; but occasionally this associated action is lost with accommodation, although the light-reflex remains. The external muscles of the eyeball are also sometimes affected. As the ciliary muscle is regaining power there may be undue convergence on accommodation from the increased voluntary effort affecting the internal recti in undue degree. The most common palsy is weakness of the internal recti conjoined with the paralysis of the ciliary muscle, so that the eyes tend to diverge, just as they often do in near-sighted persons who do not use accommodation. Occasionally there is paralysis of the external recti. I have once seen double ptosis, and paralysis of all the muscles supplied by one third nerve has been observed. In two cases, almost all the muscles of both eyes became paralysed. In one the right eye was quite motionless, and in the left the only action was in the external rectus; the eyelids also drooped: in the limbs there was ataxy and some loss of power. The ocular palsy lasted a month (Uhthoff). In the other case, all the ocular muscles of both sides were paralysed and the fields of vision were restricted. There was also paralysis of palate, right side of the face, and muscles of the neck, and ataxy of the limbs. The patient died; the morbid appearances will be mentioned presently.† I have also seen a patient who described, as part of the palsy after diphtheria at the age of twenty-two, double vision and double ptosis. In four cases with paralysis of some ocular muscles, described by Morton, these came on four to seven weeks after the diphtheria, and lasted four to twenty-six weeks. Sometimes there is slight paralysis of one or another of the ocular muscles, varying from day to day.

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* See Schmidt-Rimpler, 'Berlin. klin. Wochenschr.,' 1884, No. 7.

† Mendel. For the reference to these cases see p. 190.

There is not often any other obvious affection of sight than that which results from the paralysis of accommodation. Nevertheless it is probable that a concentric contraction of the fields of vision is not uncommon; it is, as it were, masked by the effect of the cycloplegia, and so generally escapes notice. An example of it has been published by Jessop,* and it was present in the case of ophthalmoplegia mentioned above. It was found by Herschel in no less than five out of thirteen cases of diphtheritic cycloplegia examined.† No morbid change has been observed with the ophthalmoscope.‡

Of the other special senses, hearing is seldom impaired, although a case of transient complete deafness has been recorded by Tooth.§ I have twice known the sense of taste to be lost, and in one instance smell was impaired, although taste was normal.

Limbs.—The affection of the limbs succeeds that of the soft palate. The loss of motor power always comes on gradually, and varies much in degree, but is rarely or never absolute. It is usually bilateral and symmetrical; one side may be affected more than the other, but there is never limited hemiplegic weakness. The legs commonly suffer before the arms. The muscles become flabby and toneless; the common loss of the knee-jerk has been already mentioned. The weakness may merely be sufficient to render the patient unduly fatigued with slight exertion, or it may be such that standing is impossible. When considerable, there is a peculiar gait that is very suggestive; the legs are moved as if they were at once heavy and limp, and there is not the aspect of stiffness so often seen in spinal paraplegia. If the affection is slight there may be no change in electric irritability, but when considerable weakness has existed for two or three weeks there is generally a diminution of irritability to faradisation, which may proceed to almost complete loss. The muscles still respond to the voltaic current, and their reaction to it may be altered as in ordinary peripheral paralysis (see vol. i, p. 54). The irritability of the nerve-trunks is lessened to both faradism and voltaism. Superficial reflex action is also diminished or lost.

In slight cases sensation may be unaffected, but it usually suffers when the motor paralysis is severe, and occasionally it is impaired when there is little motor weakness. Subjective sensations of "numbness," tingling, "pins and needles," formication, &c., and sometimes hyperæsthesia, may precede diminution of sensibility, and are often complained of when there is no actual loss. When this exists it may involve equally pain and touch, or tactile sensibility alone; it is always greatest towards the extremity of the limbs. I have known anæsthesia to be confined to the fingers and thumb of one hand, in a case in which both tactile and painful sensibility had been lost over both legs. Sometimes the distribution of anæsthesia is peculiar; the tips of the fingers may be alone affected. In one patient, tactile sensibility was lost on the palms and soles and preserved elsewhere, even on the dorsal surfaces of the hands and feet. In one case the anæsthesia of the extremities extended to the middle of one, and the upper part of the other forearm, and to the middle of each thigh.* If there have been pains in the limbs, the loss of sensation may correspond to them in position. Muscular sensibility may also be impaired.

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* 'Trans. Ophth. Soc.,' 1886.

† 'Berlin. klin. Wochenschrift,' 1883, No. 30.

‡ Care must be taken not to mistake for this the hysterical amblyopia which, in girls, sometimes comes on during diphtheritic paralysis (se

§ 'Brit. Med. Journ.,' 1893.

Often there is clumsiness or definite inco-ordination of movement, over and above the weakness, and this may be marked when there is little or no loss of power. In some cases it is associated with cutaneous anæsthesia, but it is certainly independent of this, since it has been observed when sensation was unaffected. The inco-ordination may involve the arms or legs or both. In the legs the disorder may closely resemble locomotor ataxy, for which it is occasionally mistaken; the anæsthesia and absent knee-jerk facilitate the error. Sometimes in the arms the disorder of movement resembles that of general paralysis of the insane—there are slight irregular twitching movements, which may render it difficult for the patient to take hold of an object.

The *trunk* muscles are less frequently affected than are those of the limbs, but they may become so weak that the patient is unable to turn over in bed. The paralysis of the intercostals may interfere seriously with respiration, and unexpelled mucus may accumulate in the bronchial tubes and give rise to alarming paroxysms of dyspnœa, which in children are often excited by emotion. Duchenne asserted that the muscles of the bronchial tubes are paralysed in these cases, but the fact rests on inadequate evidence, since there is usually sufficient weakness of the thoracic muscles to explain the symptoms. When the weakness invades the neck muscles, the head cannot be supported, and the diaphragm sometimes ceases to act. The latter is a grave symptom, and if it supervenes on, or is quickly followed by, weakness of the intercostals, death usually follows. Fortunately, the paralysis rarely reaches its height in the two sets of respiratory muscles at the same time.

Symptoms of *cardiac* failure are among the most grave, but among the less common manifestations of the disease, and are supposed to depend on paralysis of the vagus. Irregularity of the action of the

* Hallager, 'Hosp. Tid.' and 'Neur. Cent.,' 1890.

heart is not uncommon apart from graver cardiac symptoms. Of these the first indication is generally retardation of the pulse, which may afterwards become unduly rapid, and irregular or intermittent. In some cases, the retardation precedes the quickening of the pulse. In either condition, fatal syncope may occur. According to Leyden, these symptoms ought to be ascribed to the degeneration of the muscular substance of the heart rather than to true paralysis of the pneumogastric; but it is more probable that the visible changes in the cardiac tissue are the result of the nerve lesion. Dyspnœa and irregular breathing are sometimes associated with the cardiac symptoms, and have been likewise ascribed to an affection of the vagus.

The *bladder* is affected only in very severe cases. There may be either retention, with or without overflow incontinence, or there may be simple incontinence. The anal sphincter seldom suffers, but the obstinate constipation sometimes met with has been ascribed to a paralysis of the muscular coat of the intestine. Loss of sexual power is not uncommon in adult males. Symptoms of vaso-motor paralysis are never met with, nor is there any marked tendency to disturbance of the nutrition of the skin.

Complications.—Convulsions have been observed in a few very severe cases, in which the paralysis has commenced during the course of the primary disease. They are to be regarded rather as a complication of the diphtheria than as part of the paralysis, and probably result, in most cases, from an organic cerebral lesion. Hysterical paralysis, motor and sensory, occasionally complicates a true diphtheritic palsy in those who are predisposed to hysteroid disturbance. Hysterical symptoms may also succeed the specific paralysis. This complication sometimes gives rise to much difficulty in diagnosis, as will be mentioned presently.

Three other forms of palsy may be associated with diphtheria. In the course of this, as in most other acute specific diseases, sudden hemiplegia may indicate the occurrence of a vascular lesion of the brain, either thrombosis, embolism, or a hæmorrhage. Death may result, as in two of three cases mentioned by Mendel: in one of these a post-mortem examination showed a small hæmorrhage, the size of a cherry-stone, damaging the internal capsule.* The paralysis is often permanent.

Secondly, diphtheritic paralysis may be accompanied by the rapid paralysis of the whole of one cranial nerve, differing in its isolation and rapidity from the ordinary palsy, and such paralysis may be permanent. Paralysis of the muscles of mastication, so that the jaw could not be raised, followed ordinary diphtheritic paralysis in a boy of twelve, and was unchanged fourteen months afterwards.† Such an affection is probably due to an acute nuclear inflammation.

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* 'Neur. Centralbl.,' 1885, p. 133.

† Dundas Grant, Brit. Laryngol. Association, Dec., 1892.

Thirdly, some time after diphtheria, and usually after the ordinary palsy, the symptoms of slow degeneration of certain cranial nerves may develop and gradually increase during many months. In one case, weakness of the lips, tongue, and pharynx had thus developed, together with partial external and internal ophthalmoplegia and primary atrophy of the optic nerves. In each seat the symptoms became stationary before they were complete, and remained unchanged. In another case, a woman of forty, sight began to fail six months after diphtheria, and was almost lost at the end of another year. Two and a half years after the primary disease there was grey atrophy of both discs, left internal ophthalmoplegia, and weakness of some of the movements of both eyeballs, especially upwards.

Albuminuria is very common in diphtheritic paralysis, usually persisting from the primary disease. In severe cases, that are quickly fatal, it is seldom absent.

Course and Duration.—Diphtheritic paralysis varies extremely in its extent, severity, and course. Motor and sensory symptoms may lead the way. In a case very carefully observed by the sufferer, a German physician, tingling and formication were followed by anæsthesia, and this by motor weakness and ataxy, and the symptoms passed away in the opposite order.* In some cases the palsy is limited, slight, and transient. In others, region after region is successively attacked; the parts which suffer first, as the palate and eye, may recover before the limbs are involved; and when these are improving, and a speedy convalescence is hoped for, paralysis of the trunk and respiratory muscles may come on and throw the patient into extreme danger. Irregular waves of palsy seem to flow through the body, sometimes quickly, sometimes slowly, and, determined as it is by influences that we cannot discern, its course can never be foretold. As a rule, however, the palsy does not return in a part which it has once left, and when local improvement has set in it usually continues, although other parts may become weaker. It has been said that when the paralysis commences before the primary disease is over, its course from one part to another is slow and often discontinuous, but when it commences some time after the primary disease it exhibits a much slighter tendency to remission (Sauné). To this rule, however, the exceptions are very numerous. In fatal cases death often occurs early, even within the first week from the onset. Sir William Jenner has pointed out that in the most severe and serious cases the interval between the primary disease and the onset of the palsy is usually short. But the paralysis has developed rapidly and in severe degree, so as to cause death within a week, in cases in which it commenced a month after the primary disease was over.

The total duration of the paralysis, both general and local, varies according to its extent, but is generally from six to eight weeks in cases that recover. A local paralysis lasts longer if other parts are affected than if it is the only manifestation of the disease. When the palate alone is affected, it usually recovers in two or three weeks; but if the limbs also suffer, the paralysis of the palate may last a much longer time. Loss of power in the limbs always endures longer than the affection of the palate or eye; it rarely passes away in less than two months, and may last for four or six months. When definite improvement has commenced, it often proceeds rapidly, although the paralysis may have been severe and of long duration. Thus, in one case, paralysis of the limbs commenced in the seventh week, slowly increased until the twelfth week, remained almost complete until the twentieth week, when improvement commenced, and in four weeks more all trace of weakness had passed away. As power is regained, the electric irritability of the muscles becomes normal. The loss of the knee-jerk is the last symptom to pass away, and often continues for several weeks after good power has been regained. Ultimate recovery is almost always perfect.*

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* Hausemann, 'Virchow's Archiv,' Bd. cxv.

Death may occur during the course of the paralysis from other sequelæ of the primary disease, especially from renal mischief. The palsy is fatal chiefly by three mechanisms. (1) Syncope from paralysis of the heart. (2) Asphyxia from paralysis of the muscles of respiration. (3) Exhaustion in consequence of inability to take sufficient food, due to paralysis of the pharynx, or to the anorexia, and to the repugnance to food produced by the liability for it to get into the larynx. The third mechanism is effective especially in children.

PATHOLOGICAL ANATOMY.—The brain, spinal cord, and nerves present, as a rule, no morbid appearances to the naked eye beyond a variable amount of hyperæmia or minute extravasations into their substance. Very rarely larger foci of hæmorrhage are met with in the brain. In one acute case, extravasation existed between the crura, together with many minute hæmorrhages, and extensive "diapedesis" of the red corpuscles.† Such a condition suggests that the intensity of the poison had caused acute degeneration of the walls of the vessels. Meningitis, with scattered spots of exudation around the spinal cord and medulla oblongata, was found by Pierret in one case, but in this the symptoms were different from those of ordinary diphtheritic paralysis, and death occurred on the second day. In the ordinary form of palsy the membranes are always healthy, except that there may be signs of congestion, and even, in some cases, hæmorrhage about the nerve-roots.* Nor are changes found in other organs with the exception of the heart; when death occurs with symptoms of cardiac paralysis, the substance of the heart may be pale from degeneration of the muscular fibres.

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* In extremely rare cases some part, as the palate, has not recovered, and other paralyses have developed and persisted, apparently due to chronic disseminated myelitis. In one child a form of irregular bulbar paralysis (lips and palate) developed after diphtheritic paralysis at four, and persisted at eleven (Stadthagen, 'Archiv f. Kinderh.,' Bd. v). I have seen one patient in whom, a year after the diphtheria, and when all other symptoms had long ceased, there was considerable defect of power in the muscles moving the left foot, with diminution of faradic irritability.

† Kraus, 'Neur. Cent.,' 1888.

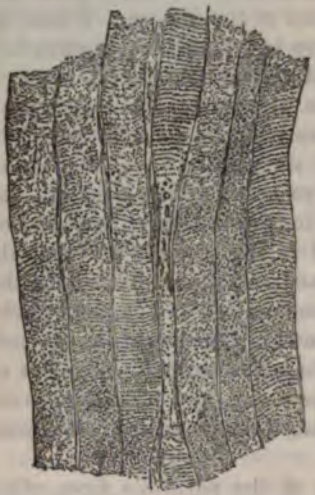

FIG. 164.—Diphtheritic paralysis. Fibres from a paralysed muscle. Recent preparation. Granular and fatty degeneration.

The true pathological anatomy of the disease is revealed only by the microscope. The muscles have sometimes been found normal; in other cases, especially those of long duration, granular and fatty degeneration of the fibres has been found, variable in degree, even in the same part of the muscle and in different parts of the same fibre (Fig. 164). This change is usually most intense in the palate, but has been found also in the muscles of the limbs. In some cases, however, notably in a series of four, presumably observed in one epidemic, the paralysed muscles presented the signs of intense parenchymatous and interstitial inflammation and degeneration; in those that were only weak the changes were slighter, while in the nerves and centres no morbid change could be detected.†

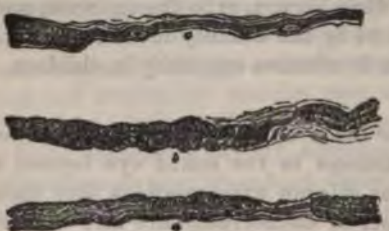

FIG. 165.—Changes in the fibres of the anterior roots, picro-carmine preparation. (After Meyer, 'Virchow's Archiv,' Bd. lxxxv.)

a. Overgrowth of the protoplasm and nuclei of the sheath; the axis-cylinder is continuous, although the medullary sheath is interrupted for a short distance.

b. Accumulation of granule masses, in places interrupting the axis-cylinder, fragments of which can be seen between the globules of myelin.

c. A fibre in which the degenerating white substance ceases suddenly, leaving the axis-cylinder only covered with the thickened sheath.

In almost all cases degeneration is found in the nerves going to the paralysed parts, either in the peripheral parts of the nerves or in their whole extent, even including the anterior roots, and, in rare cases, the posterior roots.‡ The change not only corresponds to the palsy in distribution, but is proportioned to it in

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* Buhl, 'Zeitsch. f. Biol.,' 1867, p. 359. † Hochhaus, 'Virch. Archiv,' Bd. cxxiv.

‡ Arnheim, 'A. f. Kinderheilk.,' xiii.

degree. It consists (see Fig. 165) in segmentation and breaking up of the white substance of the nerve-fibres, sometimes a multiplication of the nuclei of the sheath, and an accumulation of granule corpuscles among the remains of the fibres. The axis-cylinders usually persist, except where the destruction of the medullary sheath is most advanced, and there they may be interrupted, and in some cases they have even disappeared from the peripheral parts of many fibres. There is not, as a rule, any inflammatory change in the interstitial tissue of the nerves, and the primitive sheath is generally unchanged. The alterations are very conspicuous in the nerves of the palate, where they were discovered by Charcot and Vulpian in 1862, and to these nerves it was thought the change was confined, until it was found in the anterior spinal roots by Buhl in 1867, and in the phrenic nerve by Louville in 1872; but the wide extent of the change, and its correspondence with the position and degree of the paralysis, were only demonstrated by Déjerine in 1878.* The alteration (which can be best revealed by treating recent specimens with osmic acid) is thus limited, as a rule, to the nerve-fibres. It is essentially the same as occurs in simple degeneration of nerves, and is often described as "parenchymatous neuritis." It is the more marked the longer the paralysis has lasted. It has even been found (in one series) in all the cranial nerves, except the first, second, seventh, and eighth.† Interstitial multiplication of nuclei has been seldom observed, except in the nerves of the palate, but it was present in the case of ophthalmoplegia mentioned on a preceding page; the nerves to the eyeball muscles presented indications of interstitial inflammation, as well as the acute degenerative changes in the fibres themselves. Minute hæmorrhages were found in the sixth nerves. In one exceptional case, Meyer found nodular swellings of various nerves, formed by cellular elements which separated the nerve-fibres (Fig. 166, lowest figure).

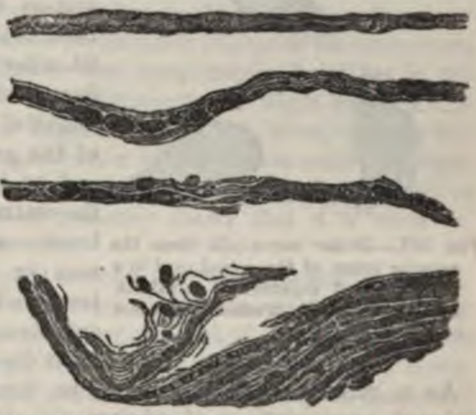

FIG. 166.—Interstitial and parenchymatous changes in the phrenic nerve: osmic acid preparations. (After Meyer, loc. cit.)

The three separate fibres show degeneration of the nerve-fibres (segmentation of the myelin, &c.), with some increase of the nuclei.

The lower group of fibres is from one of the nodular swellings on the same nerve, and shows, in addition to the degeneration of the fibres, considerable increase in the interstitial tissue.

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* Since corroborated by Meyer, Gaucher, Arnheim, Martin, and many other observers.

† Arnheim, loc. cit.

If the elements of the spinal cord are treated with osmic acid in the recent state, the motor cells are sometimes found in a state of intense granular and fatty degeneration, such as is shown in Fig. 167. In the hardened spinal cord the white columns are always normal, and in many cases no change has been found in the grey substance. In others, however, the motor nerve-cells of the anterior cornua have been found altered, either swollen, and unduly homogeneous or vitreous in aspect, or smaller than normal, and often with shrunken processes.* In rare instances collections of nuclei have been found in the grey substance. Of other changes which have been described, the significance is very doubtful; such are "rarefaction of the grey substance" (Vulpian) and an increase of nuclei around the central canal, often filling up its lumen—an appearance frequently seen in cords from healthy subjects, and which is probably without significance in this or any other disease.

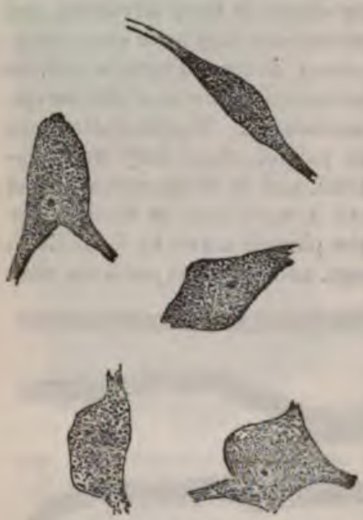

FIG. 167.—Motor nerve-cells from the anterior cornu of the spinal cord in a fatal case of diphtheritic paralysis. Recent preparation treated with osmic acid.

As a rule, no organisms can be found in the nerve-centres, although accumulations of micrococci in the vessels were described by Oertel,† and of rod-shaped bacteria by Klebs.‡ A remarkable case has been described by Buhl,§ in which the nerve-sheaths, &c., were infiltrated with bodies described as like those in the false membrane. In this case there were also numerous infarcts in the brain, as if from extensive vascular obstruction.

PATHOLOGY.—The alterations in the nerves agree perfectly with the indications afforded by the electrical reactions in severe cases.‖ There is the loss of faradic irritability which always indicates degeneration of the nerve-fibres, and the persistence of voltaic irritability which shows that the muscular tissue still preserves its contractile power. The alterations in the nutrition of the muscular fibres, sometimes found, are those that may occur in other forms of nerve degeneration and neuritis, and correspond to this also in their inconstancy and variations.* In such cases it is probable that the muscular changes are purely secondary to those of the motor nerve-fibres. But in other cases it would seem as if the cause of the palsy had acted solely on the muscles, since changes were found in these alone (see p. 912).

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* Vulpian, 'Mal. du Syst. Nerv.,' 1878; Déjerine, 'Gaz. des Hôp.,' 1880, No. 42; Abercrombie, 'Trans. Internat. Med. Congress,' 1881; Percy Kidd, 'Med.-Chir. Trans.,' 1883; Sharkey, 'Brain,' 1890. Kidd observed extensive vacuolation of the cells. The probable significance of this change has been already mentioned; it seems to occur in cells the nutrition of which is gravely altered. Kidd's figures of the vacuolated cells are almost exactly like those found by Sankey and myself in the dog in chorea ('Med.-Chir. Trans.,' 1879).

† 'Ziemssen's Handb.,' ii, 1876, p. 608.

‡ Eulenburg's 'Real-Encyclop.,' iv, p. 167. § 'Zeitsch. f. Biol.,' 1867.

‖ The significance of the electrical reactions was pointed out as long ago as 1866 by Ziemssen ('Berl. kl. Wochenschrift,' 1866, Nos. 43 and 44).

The degeneration of the nerve-fibres is certainly, in most cases, independent of any alteration of the cells of the spinal cord; it is most intense at or near the periphery, extending upwards for a variable distance, and thus it agrees with the alterations so often found in cases in which a primary degenerative neuritis has resulted from the influence of a toxic agent. But in other cases considerable change in the nerve-roots has been associated with slighter but unequivocal alterations in the motor cells of the spinal cord. A transient and slight lesion of the cells often causes a greater degeneration in the fibres, but the fact that perfect recovery and regeneration of the nerves occurs in diphtheritic paralysis, shows that its cause cannot be an actually destructive alteration, and its peripheral intensity shows that it is there independent of changes in the cells.

Thus the facts of pathological anatomy show that the motor palsy depends essentially on acute alterations in the nutrition (revealed by changes in the structure) of the lower segment of the motor path (see vol. i, p. 176), essentially "parenchymatous," beginning in the nerve-elements themselves, although vascular disturbance may be associated with it, as it so often is with various acute derangements of nutrition. But the facts also show that the three motor structures, the nerve-cells, the fibres, and the muscles, suffer in various degrees. The affection of the fibres is by far the most frequent; that of the cells is slighter, inconstant, and independent; that of the muscles is usually moderate and secondary to that of the nerves, but occasionally is primary, intense, and almost or quite isolated. To the rule that the neuritis is not interstitial, one frequent exception should be noted, that of the nerves of the palate. The palatine nerves are contiguous to the inflammation of the primary disease, and it is probable that they are influenced by the abundant growth of organisms in the false membrane, which often penetrate into the substance of the mucous membrane, and even into the tissue beneath. Interstitial inflammation may ascend nerves, although the purely parenchymatous neuritis only descends them; hence it is not surprising that the palatine nerves should sometimes present evidence of inflammation through a considerable extent. Their affection was long held to be the mechanism by which the whole of the nervous lesion was produced. It was thought that the inflammation ascends these nerves to the centre, and there spreads. But the discovery of the lesion, and the demonstration that it is as discontinuous as the palsy, have rendered the theory chiefly of historical interest, although it has been even lately maintained that palatine anæsthesia may be caused by the compression of the nerves by inflammatory products.

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* Occasionally in injuries to nerves or in disease of the anterior grey matter of the cord, the muscular fibres present very intense granular and fatty degeneration.

The special susceptibility of the palate is not, however, altogether explained by the fact that the local inflammation may extend to the sheaths of the palatine nerves. A considerable time usually intervenes between the primary angina and the palsy of the palate, and the latter may be the first symptom of the palsy that follows diphtheria of a surface wound, when the throat affection is altogether wanting. From this it is evident that there is a special susceptibility of the centres or nerves of the palate. If this is true, it becomes a question whether the local inflammation has as much to do with the occurrence of palsy of the palate as the state of its nerves would suggest. The peculiar influence of some poisons on certain parts of the nerve-centres is well known, and is illustrated in this disease by the paralysis of accommodation—a phenomenon as remarkable, if not more remarkable, than the paralysis of the palate. The lesion underlying the cycloplegia has not yet been discovered, and we do not know whether it is central or peripheral. In the association of the changes in the medulla, in hydrophobia, with the affinity of the virus for the salivary gland, we have a peculiar and significant central and peripheral correspondence.

The impairment of sensation is certainly due, in many, and probably in most cases, to the degeneration of the sensory nerve-fibres. Whether an affection of the nerve-cells of the cord, in which the fibres end, co-operates in producing the symptom we do not know. It is not improbable, but the evidence on which such a central cause of anæsthesia has been assumed (even recently) is invalid. It is the symmetry and limitation of the loss, in such cases as that in which it involved only the palms and soles. Peripheral fibres possess a susceptibility related to their position, which, alike mysterious and certain, gives rise to a distribution of symptoms suggestive of a central disorder. It is possible, indeed, that, as is certainly the case with the motor structures, the central and peripheral sensory elements may possess a corresponding local instability of resistance to the action of a special virus.

The isolated loss of the knee-jerk may be due to impairment of the motor cells or fibres; when there is actual palsy, the motor lesion affords an ample explanation of the loss, since the symptom would certainly result from the structural changes discovered in these cases. An alternative explanation of the isolated loss is that there is an affection of the sensory muscle-nerves analogous to that which probably causes the symptom in tabes (see vol. i, p. 428). Distinct muscular anæsthesia has been observed in diphtheritic palsy, and an affection of these nerves, or of their central cells, is the probable explanation of the ataxy that is sometimes so conspicuous. According to this theory, the resemblance between post-diphtheritic ataxy and the condition of the tabetic patient is more than superficial, since the inco-ordination of tabes is chiefly due to an affection of these nerves.

The relation of diphtheritic paralysis to the primary disease is a subject on which recent investigations have thrown much light, direct and indirect. Such changes as underlie the symptoms have always been found, when their cause could be ascertained, to be due to the action, on the structures, of a poison circulating in the blood. It is thus brought into contact with the nerves, of which some are especially susceptible to its influence. Of this many instances have been adduced ("Multiple Neuritis," in vol. i). The poison seems to be, as a rule, a chemical substance, and, when due to a specific disease, is produced by the organisms, directly or indirectly. Very clear proof of this has been furnished in the case of tetanus, and a similar, if less complete proof, in the case of diphtheria, has been supplied by the important investigations of Martin.* He finds that an albuminose is present in the blood in diphtheria which, injected into rabbits, causes not only the same symptoms, but also the same nerve lesions as are met with in diphtheritic palsy in man. He believes that it is not due directly to the bacilli of diphtheria, but that these produce a ferment, which acting on proteids in the tissues, especially in the spleen, convert them into the toxic albuminose. It is probable, however, that more facts have yet to be discovered regarding the "toxine." The differences in the structure that suffers in different cases of paralysis, and perhaps in different epidemics, suggest that the material varies in precise character and action, and that more than one may be formed, as in the case of tetanus.† Moreover other varieties in the agents may occasionally exist, and may explain such facts as the remarkable series recorded by Boissarie.‡ In a certain district of Paris there occurred a series of cases of severe diphtheria, and at the same time a series of cases of paralysis of the palate, eyes, limbs, heart, &c., perfectly like that which occurs after diphtheria, and accompanied by albuminuria. The remarkable fact is that in these cases of primary palsy, there was no history of preceding sore throat, and in several of the cases distinct diphtheria followed the paralysis, which lessened during the throat affection. Some of the cases of primary palsy seemed distinctly to arise by infection.

Diagnosis.—A definite attack of diphtheria, and the characteristic onset of the subsequent paralysis, render the diagnosis as a rule a

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[*] S. Martin, 'Gulstonian Lectures,' 1892.

[†] Martin assumes that the bacilli of diphtheria do not enter the blood, but organisms have been occasionally found, as we have seen, in the nerve-structures.

[‡] 'Gaz. Hebd.,' 1881, Nos. 20 and 21.

simple matter. It is only when the nature of the preceding disease has escaped recognition that any real difficulty occurs. It must be remembered that an offensive discharge from the nose is sometimes the chief indication of diphtheria. But the onset by an affection of the throat and eye, and the subsequent progressive paralysis in the limbs, are so distinctive as to indicate at once the nature of the symptoms, and often also that of the preceding sore throat. In most cases of this character in which a mistake is made there has been a considerable interval between the throat affection and the palsy.* Greater difficulty is presented by those cases in which little attention has been paid, not only to the primary disease, but also to the early symptoms, which may pass away before the limbs become affected. In such a case the later symptoms may be ascribed to a primary disease of the spinal cord. The absent knee-jerk and inco-ordination may simulate locomotor ataxy, or the weakness may be mistaken for simple paraplegia, or the wasting be ascribed to a subacute polio-myelitis. An important distinctive feature is the manner in which the weakness spreads slowly from one part to another, often lessening in the part first attacked. In such cases careful inquiry will generally elicit a history of transient difficulty in swallowing and in reading, commencing not long after a sore throat. The change in electrical irritability is usually much slighter than in acute atrophic paralysis, while in the latter there is no affection of sensation, and there is a more sudden onset. The onset of diphtheritic paralysis is more rapid than is common in ataxy, from which also the marked weakness and the absence of much pain help to distinguish it. In simple spinal paraplegia the knee-jerk is usually excessive, and it is never absent unless there is marked wasting or anæsthesia in the thigh.

It is not often that there is any difficulty in distinguishing between diphtheritic paralysis and hysteria. The affection of the palate, almost invariable in the former, is never met with in the latter disease. A difficulty is sometimes occasioned by the fact that a true diphtheritic paralysis may be succeeded by hysterical palsy or accompanied by other hysterical symptoms. Thus, in one case, paralysis of the palate and ciliary muscle had ceased, but was succeeded by general loss of power and of sensation, supposed also to be diphtheritic. The girl was lying in bed almost motionless, with universal anæsthesia and convergent strabismus from, it was assumed, paralysis of both sixth nerves. Under emotional excitement, however, the limbs could be moved with considerable force. The anæsthesia was universal in extent, a most unusual symptom in diphtheritic paralysis. Moreover, the eyes could be moved to right and left, but the convergence was maintained during the movement and was thus clearly due to considerable spasm of the internal recti; all the symptoms passed away after a few days' "moral treatment." In such cases the knee-jerk may give important guidance. Thus a girl presented herself with weakness of the left leg, and anæsthesia of both leg and arm, symptoms that had existed since an attack of diphtheria six months before. There was also loss of accommodation and of convergence. The latter were no doubt diphtheritic, but the knee-jerk in the leg was normal, and this made it probable that the weakness and the anæsthesia were purely functional—an opinion confirmed by the previous history of the patient and the subsequent progress of the case. She had had a similar transient hemiplegic attack two years before, and faradism removed the palsy in a few days.

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* Thus a young man came to me with distinct symptoms of diphtheritic paralysis, the nature of which had not been recognised. He had lost a brother from diphtheria three months before, and the patient himself had suffered from a sore throat ten days before his brother was taken ill. He had not seen a doctor, considering that such affections "were better left alone."

The PROGNOSIS in diphtheritic paralysis is, on the whole, good in cases of deliberate onset, so long as there is not grave exhaustion from the preceding disease, evidence of cardiac paralysis, weakness of the muscles of respiration, or inability to take food. In each of these conditions there is danger. Considerable retardation in the heart's action is also a very serious symptom, and so is extreme frequency or irregularity. The failure of respiratory power is most grave when both the intercostals and the diaphragm become weak at the same time; the amount of mucus accumulating in the chest is an accurate indication of the degree of danger. Paralysis of the vocal cords is rarely a cause of death, because it is usually incomplete, but paralysis in the region of the superior laryngeal nerve is serious, because it is so often associated with pharyngeal paralysis; food readily enters the insensitive opening to the larynx, and may thus cause great distress, and even reach the lungs and set up a form of pneumonia. Paralysis of the muscles of the neck is a rather grave symptom, because paralysis of the diaphragm and pharynx so often occur in conjunction with it. The sooner the paralysis comes on after the primary disease, and the more rapidly it develops to severe degree, the greater is the danger to life.

The duration of an attack is difficult to foretell. As a rule, the more severe the palsy, the longer will it last. If the paralysis of the palate is considerable and lasts some weeks, it is improbable that the limbs will escape, or that the attack will be over in less than two months. If the legs become very feeble, the arms will almost certainly suffer also. On the other hand, if the paralysis of the palate is brief in duration there is a fair prospect that the limbs may escape. It must, however, be remembered that complete recovery from the early initial symptoms does not preclude the occurrence of others, but it is not probable that, in such a case, they will be severe. Loss of faradic irritability in any region is proof that the palsy there will last for many weeks; and if to this is added the loss of voltaic irritability in many muscles the danger to life is considerable, because the poison is intense.

The prognosis is more serious in children than in adults, chiefly on account of their smaller reserve of strength, and the greater difficulty of feeding them if complications interfere with the ordinary method.

TREATMENT.—Of first importance in the treatment of diphtheritic paralysis is restoration and maintenance of the patient's strength, already weakened by the previous disease. For this end feeding is at once the most important and the most difficult measure. Easily digested nourishment should be given at frequent intervals, with wine or brandy if the pulse indicates it. When the palate is paralysed, pulpy foods are swallowed much better than liquids, and the affection of the palate does not usually interfere with a due amount being taken. A much more serious obstacle is the paralysis of the pharynx or of the epiglottis and upper part of the larynx. In the former, deglutition may be difficult or impossible; in the latter, food cannot be kept out of the larynx, and attempts to swallow occasion so much distress that mental repugnance is added to the physical difficulty, and it may be impossible to give food in the ordinary way. It is, indeed, inadvisable to do so if particles of food and drink constantly get into the larynx, on account of the danger of pulmonary complication. Food must therefore be injected into the rectum, or given by means of a small œsophageal tube or large gum-elastic catheter. A flexible tube passed through the nose does not answer so well in these cases, because the paralysis often extends to the lower part of the pharynx, and it is absolutely necessary that the extremity of the tube should be below the paralysed portion. Which method, rectal or œsophageal, should be adopted must depend on age and condition, but it is essential that the patient should not be allowed to pass more than twelve hours without food being given, in sufficient quantity, by one or the other method. It is unwise, in the case of children especially, to wait, in the hope that the child may presently be induced to take food. Few cases of severe diphtheritic paralysis can survive forty-eight hours' complete fasting, and the danger of producing exhaustion by the procedure of forced feeding is greater the longer the abstinence has continued.

The variable course of diphtheritic paralysis, and its ultimate tendency to recovery, render the influence of drugs upon it very difficult to ascertain. There is no evidence, at present, that any agent has a considerable influence on the morbid process itself. We have at least no means of rendering inert the cause of the palsy. Tonics, especially iron and quinine, often seem to be useful. Strychnine has been largely employed, and sometimes seems of actual service, but it is certainly powerless to neutralise the morbid process in its early stages, and seems to be without influence on the spread of the disease. Moreover, it is not wise to give large doses of a drug that stimulates so powerfully the affected nerve-cells. Recovery has been ascribed to its hypodermic use, but the evidence of a relation between the two has in published cases, been no more distinct than when the drug has been given by the mouth. Belladonna in large and frequent doses has been thought by Abercrombie to do good.

Of local treatment the most important measures are rubbing and electricity. The slowly interrupted voltaic current should be used, to which alone the muscular fibres are capable of responding if the paralysis is severe. The current should, if possible, be of such a strength as will cause the muscles to contract, but, in the case of children, if an adequate strength causes severe emotional disturbance, it is far better to be content with a weaker current, as strong as can be borne without distress or alarm. The utmost good electricity can do, is, in this disease, extremely small compared with the harmful influence of a daily fright.

Among special symptoms which may require treatment, the paralysis of the pharynx has been already considered. The loss of accommodation scarcely calls for treatment, and it is doubtful whether anything can be done for it, although the application of eserine (a half per cent. solution in water) twice a day, has been recommended by Herschel. If there are symptoms of cardiac failure, the patient should be kept in the recumbent posture, since fatal syncope has occurred on sudden rising. For undue frequency of action, small doses of digitalis may be given, the effect being carefully watched. Duchenne recommends faradism to the precordial region as a powerful means of stimulating a failing heart, and several other French writers have endorsed his recommendation.

Failure of the muscles of respiration is rarely so complete as to cause death directly, but when mucus accumulates in the chest, suffocating paroxysms are apt to occur, which are attended with some danger. If life is threatened in one of these, artificial respiration may carry the patient over the attack, and Duchenne advised reflex stimulation of the respiratory centre by faradism to the back of the chest. An account of a case in which the method was employed with success has been given by Millard.* The patient was a man aged thirty, and paralysis of the palate, diplopia, and weakness of the limbs had existed for about three weeks, when distress in breathing suddenly came on; expiration was short and sudden; mucus accumulated in the air-passages, causing a loud tracheal râle; the patient could not expel it, and asphyxia seemed imminent. Duchenne was called in, and, noting that the diaphragm acted well, and that the source of the trouble was the deficient expiration, faradised the skin at the back of the thorax, and thus "rapidly re-established the expiratory power, and caused the immediate expulsion of a quantity of bronchial mucus. The patient was immediately relieved, and, the application being repeated during the next few days, all alarming symptoms passed away, and he ultimately recovered."

* Quoted by Landouzy, loc. cit., p. 87.

HYDROPHOBIA.

Hydrophobia is the name given to a malady, when it affects man, which is called rabies when an animal is the subject of it. It is a specific disease, always due to a specific poison, always contracted from an animal, and, like syphilis, transmissible only by inoculation. It is manifested by symptoms that are chiefly due to disturbance of the nervous system, and it is almost always fatal.

RABIES IN ANIMALS.

Before describing the disease as it occurs in man, it will be well to give a brief outline of the characters of the affection in animals. The fullest description of it is that given by Fleming in his work on the disease.* The malady is almost invariably due to inoculation by a bite. It is the opinion of Fleming that it arises spontaneously in very rare instances, but this is not certain.† It occurs chiefly in dogs, wolves, foxes, and cats, and by these it is commonly transmitted, especially by the first two. It may also be produced in the herbivora and some rodents, but it is probably very seldom transmitted by them. In the dog the first symptom is dulness, with an indisposition to move. Then the animal becomes shy and suspicious or threatening in manner, and afterwards irritable and restless, with a strong tendency to bite. Throughout the early period there is a depraved appetite; ordinary food is rejected, and hay, straw, cloth, wood, and cinders, and even the animal's own hair, are eaten. This is an important characteristic, because the presence of such substances in the stomach constitutes one of the signs by which the disease can be recognised after death.‡ The bark usually becomes changed into a peculiar howl, which begins with a short low note and ends with a long higher note; it has also a peculiar metallic ring. There may be drooping of the lower jaw and weakness of the muscles of deglutition; this may prevent swallowing, but there is no dread of water. Ultimately, palsy always comes on; it is first manifested by unsteadiness of movement, then by increasing weakness of the legs, and at last there is inability to stand. The animal then usually becomes comatose and dies. A distinction has been made between "dumb or paralytic rabies" and "furious rabies," but the difference depends only on the degree of excitement; all cases, if permitted to run their course, end in paralysis, but in the "paralytic" form this is an early symptom, and the stage of excitement may be represented only by quickened breathing.* After death, distinct changes are often to be found in the nerve-centres. There is congestion of the membranes and in the cortex, with accumulations of lymphoid cells about the vessels, especially intense beneath the lower part of the fourth ventricle, where I have found the tissue in a state indistinguishable from that of acute inflammation. The changes are essentially the same as those to be described as often present in man. There may be minute extravasations, and sometimes hæmorrhage visible to the naked eye. Similar but usually slighter changes are often found in the grey matter of the spinal cord. The larynx and trachea are usually congested, and so also are the lungs.

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* Some additional facts, observed during a long series of investigations, are described by Dowdeswell ('Proc. Royal Society,' vol. xliii, p. 48). His description has been chiefly followed in the above account.

† See p. 925.

‡ This sign has, indeed, been regarded as pathognomonic, but doubts have been thrown on its value. Gibier ('Gaz. Heb.,' 1884, No. 29, &c.) states that he has repeatedly failed to obtain evidence of the rabid state of animals in whose stomach hay and straw were found, when he employed the test of subdural inoculation. Mr. E. Batt, Vet. Surgeon to the Brown Institution, informs me that this feature is not uncommon apart from rabies, and that it is very difficult to feel confident, from post-mortem signs alone, that an animal has died from this disease.

In the stomach, besides the peculiar contents already mentioned, minute extravasations are commonly seen in the mucous membrane. The salivary glands present no constant changes, nor do other organs.

In the rabbit, according to Dowdeswell, the symptoms are dulness, followed by excitement, transient pyrexia, and progressive paralysis, which is the cause of death. The post-mortem changes are similar to those in the dog, but the stomach contains ordinary food.

Pasteur has discovered that the virus exists, after death, in the central nervous system as well as in the salivary glands, and Dowdeswell has found that it is also abundant in the peripheral nerves. Inoculation with the tissue of the brain and spinal cord produces the disease even more certainly than does that of the salivary glands. Pasteur has also proved that, in animals, the disease is produced far more surely, and with a more uniform incubation period, when the inoculation is made beneath the dura mater, by trephining the skull, than when it is beneath the skin.

The period of incubation is very variable in animals, especially when the inoculation is subcutaneous. In intra-cranial inoculation it is generally from seventeen to nineteen days, and this whatever is the source of the virus. By passing the virus through a series of rabbits, the period becomes shortened to six or seven days, and remarkably uniform—the "fixed virus" of Pasteur. The poison exists in the tissues only towards the end of the period of incubation. The nature of the virus has not yet been demonstrated, but that it is a micro-organism scarcely admits of doubt.† Its activity appears to be rapidly destroyed by decomposition, yet it has been found very active in a dog that had been buried fourteen days.[*] It is certain, also, that the saliva, dried before decomposition, retains its virulence for many days. In the dried spinal cord the virus gradually loses its power, which becomes extinct at the end of ten days, even in the case of the "fixed" virus of the rabbit. The pathology of the disease will be again considered.

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* Ferrée, 'Compt. rend.,' cvi, No. 12.

† Gibier ('Comptes rend.,' 1884, vol. xcviii, and 'Thèse de Paris,' 1884) and also Dowdeswell (loc. cit.) found an organism in abundance in the central nervous system in some cases; it was extremely difficult to stain and recognise, and this, if it was really the microbe of rabies, explains the frequent failure to discover it. Dowdes-

Rabies in Man.

The disease in man is commonly called "hydrophobia," because the difficulty in swallowing induces a sort of reflected mental dread of water. Although this dread may be absent, and the name at best designates only one symptom, it is so firmly established that an attempt to change it would be futile. The features of the disease are occasionally simulated by other maladies, and this fact has been made, by certain lay writers, the ground for denying the occurrence of rabies in man; but such denial is only possible through ignorance, and its refutation is needless.

In man, the disease is invariably acquired from a rabid animal, and is almost invariably due to inoculation with the saliva by means of a bite. In nine tenths of the cases the disease is contracted from a dog, in a few cases from cats, very seldom from a fox or a wolf. It has been due to a wound received in the dissection of a rabid animal,[†] but it is not certain that inoculation with the blood alone will produce the disease.

A bite is more effective if upon an uncovered part of the body, as the face or hand, than if inflicted through the clothes, by which the saliva may be wiped from the teeth. Children are often bitten about the face, and a large proportion of those who are so bitten are infected. It is said that a bite inflicted immediately after a preceding bite is less likely to infect, as the teeth may have been cleaned by the preceding bite. The disease may also be caused by a rabid dog merely licking a scratch upon the hand (and may be thus produced more often than remembered and recorded facts suggest), and it has resulted from the teeth having been used to loosen a knot upon a rope with which a rabid dog has been tied up. A person has been inoculated by a bite from a healthy dog, inflicted immediately after it had been fighting with a rabid animal, the saliva of which was no doubt inoculated. The malady has followed the scratch of a cat, but probably by the animal's saliva having thus been introduced.

It has been said that hydrophobia has been produced by the bite of

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well obtained some cultivations from it, and one animal inoculated with the cultivated organism seemed to be protected from the effects of the active virus ('Lancet,' 1886, vol. i, p. 1112).

[*] Galtier, 'Comptes rend.,' cvi, 1888.

[†] For an instance, see Bollinger, 'Ziemssen's Handbuch,' Bd. iii, p. 542.

an animal not suffering from rabies. This is opposed to all that we know of the origin of other diseases. The statement rests upon some facts which seem to establish that hydrophobia may result from the bite of a dog which did not at the time, or for several weeks afterwards, present the recognised symptoms of the disorder. The best explanation of these cases is probably that, in some rare and exceptional circumstances, rabies may affect a dog as a malady that is transient and insignificant, but communicable. Examples of this are seen in most infectious diseases. The fact that dogs may recover from rabies,[*] makes it probable that the disease is sometimes slight.

The facts mentioned show that only some of those who are bitten by a rabid animal contract the disease. The difficulty of ascertaining the proportion is great, on account of the frequent uncertainty whether the dog was really rabid. It has been calculated that, of all those bitten by certainly rabid dogs, 47 per cent. suffer; of those whose wounds were uncauterised, 83 per cent.; of those promptly cauterised, only 33 per cent.[†] On the other hand, of persons bitten by dogs merely suspected to be mad, only 8 per cent. suffer.[‡] The immunity of some persons is perhaps due to bites having been inflicted through the clothes, or to the animal's teeth having been otherwise freed from saliva. The poison may also vary in virulence at different stages.

The general incidence of rabies can be traced to the exposure to the risk of bites, their character and position. Hence more males than females suffer, the proportion being about four to one. Most males are in the middle period of life. Very few adult women are affected, but children of both sexes often suffer; they are much exposed to the risk of bites on the face and hand from straying dogs. No less then two fifths of all cases are under fifteen.[§]

The period of "incubation" varies between wide limits, and is longer and more variable than that of any other known acute specific disease. The common period is from six to ten weeks. Bauer has calculated the average period of 510 cases to have been seventy-two days, and rather longer in men (eighty-five days) than in women (sixty-five days). It is shorter when the bite is on the head or neck than when on the limbs.* In at least half the cases, the disease develops in from one to three months after infection. In a few cases, the period is less than a month, the shortest observed having been about twelve days. Cases are not uncommon in which the period is more than three months—six, nine, and in some cases even twelve or eighteen.† Instances have, indeed, been recorded in which five, ten, or twelve years are said to have passed; most authorities consider that in such cases there has probably been a second unnoticed infection. It is, however, certain that the disease may occur after an interval of a year or of eighteen months, and it is difficult, therefore, to deny the possibility of a longer interval. Although it is common to speak of the interval as the "incubation" period, it is probable that only the shorter periods really correspond to what we understand by the term in the case of other diseases.

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[*] Observed by Pasteur and Azary. The latter proved the disease to be rabies by inoculation from the body of the person who was bitten and died ("Buda-Pest Comm. Rep.," by Laufenhain, 'Cent. f. Nervenkr.,' 1889, p. 293).

[†] We need more facts as to the actual interval before cauterisation. Many persons suffer whose wounds are cauterised in a quarter of an hour—ample time for the virus to pass beyond the reach of caustics. But it is doubtful whether the disease ever occurs if a stick of nitrate of silver is immediately plunged into a small wound.

[‡] Bollinger, loc. cit., from the statistics of Tardieu, Thambayn, and Bouley.

[§] During the twenty-five years ending 1872 ('Reg.-Gen. Rep.,' 1875), 299 males and 74 females died from hydrophobia. 89 males and 33 females were under 15, = one third of the males, and four sevenths of the females. The greater relative male liability is least (but still considerable) in childhood, and increases up to 45; the ratio between the sexes is 2 to 1 in the first five years, and 3 to 1 in the third; during the thirty years 15—45 it is 8½ to 1. In later life the preponderance of males is not so great, but the cases are few.

Symptoms.—During the interval after the bite there are commonly no symptoms. Occasionally, towards the end of the period, pain or unpleasant sensations are felt at the seat of the wound, explicable in part (but not altogether) by the attention which is directed to it. Mental depression is occasionally conspicuous, but is commonly the result of the sufferer's knowledge and anticipation of the possible consequences of the bite. At the onset of the acute symptoms there may be considerable local pain, occasionally radiating up the limb. It may exist for a few days or a week before other symptoms. Even more frequently, all local symptoms are absent. Slight preceding fever (even with a rigor) has sometimes been observed for a day or two, but usually the first indications of the impending malady are a sense of general malaise, mental depression, and disturbed sleep. These may exist for a few days, even for a week or more, before the actual onset, which is indicated by some discomfort about the throat, an occasional sense of choking, or a little difficulty in swallowing liquids. The effort, if successful, has been aptly compared to that of a child taking a nauseous draught (Handford). The spasm in the pharynx, produced by the attempt, increases in the course of a few hours, and spreads to the muscles of respiration, causing a short, quick inspiration, a "catch in the breath," resembling that which is produced in health by an affusion of water. This soon becomes a strong inspiratory effort, in which the "extraordinary muscles of respiration," sterno-mastoid, scaleni, &c., and even the facial muscles, take part; the shoulders are raised, and the angles of the mouth drawn outwards. Very rarely sudden severe throat-spasm has been the first symptom.

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* Bauer, 'Münch. med. Wochensch.,' 1886. If 27 doubtful cases of alleged very long incubation were included, he found the average of 537 cases to be 126 days. The period presents very little variation in the case of bites by different animals.

† Many well-authenticated instances of eighteen months' duration are on record.

As the intensity of the spasm increases, so does the readiness with which it is excited. It may be caused by the mere contact of water with the lips, and a state of cutaneous hyperæsthesia develops, so that various impressions, such as a draught of air, which normally excite a respiratory effort, bring on the spasm. The mere movement of air caused by raising the bedclothes may be sufficient. The patient is often unable to swallow the saliva, which is usually abundant and viscid, so that it hangs about the mouth and is expelled with difficulty; this greatly increases the patient's discomfort. Vomiting is common, a greenish-brown liquid being ejected. The attacks of spasm are very distressing to the patient; the mental state which they occasion increases the readiness with which they are produced, and in some cases the mere sight of water or the sound of dropping water will cause an attack. It may even be excited by visual impressions which cause a similar sensation, as the reflection from a looking-glass, or even a strong light. The sufferer's horror and dread of these excitants becomes intense. Thus the disturbance in the act of swallowing liquids, which constitutes as it were the first symptom and key-note of the disease, spreads, on the one hand, to mental disturbance, and on the other to extensive muscular spasm.

In each of these directions further symptoms develop. The spasm, at first confined to the muscles of deglutition and respiration, spreads to the other muscles of the body, and the paroxysms, at first respiratory, afterwards become general, and assume a convulsive character, although still excited by the same causes. The convulsions may consist of general muscular rigidity, sometimes tetanoid in character, with actual opisthotonos, or they may be co-ordinated and closely resemble hysteroid convulsions. During this stage the knee-jerk is commonly increased and also reflex action from the skin. All the cerebral centres may share the over-action, and auditory hyperæsthesia is sometimes very distressing. In other cases the mental distress passes into delirious disturbance, in which, at times, the balance of reason is lost, and the distress gets command over the mind. The horror with which the attempts to drink and the causes of the distress are regarded, becomes transferred to the attendants by whom they may have been produced. Actual delusions may supervene, and may even pass into a state of positive mania. The mental derangement is most intense during the paroxysms of spasm, and the frenzied patient may spit his saliva at those about him, and often attempts to bite them with his teeth, making occasionally strange noises in his throat which have been thought to resemble the barking of a dog. The sight of a dog has been known greatly to intensify the mental excitement, and this, strangely enough, in cases in which the sufferer had no suspicion of the nature of this affection. It often happens, however, that the very anxiety itself deters the bitten person from alluding to its cause, until his mental processes are exposed by the withdrawal of the previous control. The idea of a dog may enter into the delusions in strange ways. One patient will think a dog is under the bed, or that he sees one in the room. Another is seized with so intense a fear of being mistaken for a dog that at last he fancies he is one, and goes on "all fours." But there is much that is mysterious in these delusions, as will be pointed out in the section on Pathology.

The mental disturbance is not always thus subordinate, as it were, to the other symptoms. There may be a variable amount of mental derangement almost from the first,—an unnatural suspicion, with manifestations of ill temper, succeeeded by a curious mental restlessness and loquacity, and some incoherence in the sequence of ideas. Delusions and hallucinations are usually late symptoms, but occasionally some false ideas are manifested early in the affection. Throughout its course there are sudden variations in the mental state, which are almost characteristic of the disease; a complaint, perhaps couched in indignant language, is succeeded by an apology, and the frenzied patient may alternately attempt to bite his attendants and beseech them to keep out of his way, or to hold him fast that he may not hurt them. Similar mental disturbance may be present in children, although its details necessarily differ, but in them it is especially common for initial fear of liquids to be lost in early delirium.

In adults also, as the mental disturbance increases, the respiratory spasm and difficulty in swallowing often lessen, and sometimes cease. The convulsive attacks may also cease. The patient may die, apparently exhausted by the attacks of fury, or, if life is prolonged long enough, the mental and muscular excitement may give place to a state of paralytic exhaustion, which seems to represent the paralysis that is so prominent in animals. Coma may come on, but the patient usually dies within a few hours of the diminution in the symptoms of excitement. Death, however, sometimes occurs earlier, from asphyxia during a violent paroxysm of respiratory spasm, or from sudden failure of the action of the heart, a result that is explained by the lesions found in the medulla oblongata.

Among occasional symptoms priapism deserves mention; it is not very common, but has attracted attention from ancient times.

The temperature is almost always raised. At the outset the elevation is trifling (occasionally, indeed, absent), and throughout the disease it may remain moderate, 100° or 101°. More frequently, as the symptoms increase, so does the pyrexia, and it amounts to 103°, 104°, or 105°, and may even reach a still greater height just before death, and may continue to rise for a short time after death. A rectal temperature of 108·8° has been observed twenty minutes after death (Handford). The urine very often contains albumen, sometimes as much as a quarter or a third. Sugar has been found in a few cases. The scar of the bite is said sometimes to become livid in the course of the disease.

The duration of the disease has varied in fatal cases from twelve hours to ten days. The usual duration is from two to four days; one tenth of the patients die in the first twenty-four hours, three fifths die within three days. Cases exceeding four days in duration are rare.

The course of the disease has been divided into two stages, the first distinguished by the respiratory spasm, the second by the mental disturbance and convulsions. By others, an initial stage of depression has been distinguished from the later stage of excitement. The final exhaustion is also regarded as a third or paralytic stage. But these stages are often not well marked; the elements that distinguish them may be combined in various degrees, and those of the later period are sometimes conspicuous from the first.

The special predominance of certain symptoms sometimes impresses a definite character on the attack, so that certain varieties may be distinguished. Their recognition is of some practical value, since it may prevent some of the frequent diagnostic doubts. The most important of these varieties depend on the predominance of mental symptoms on the one hand, and of convulsions on the other. Delirious and mental excitement may exist from the onset of the affection, and in such cases the respiratory spasm may be less pronounced in the early period than is usually the case. The special character of the mental derangement varies greatly according to the mental condition of the patient, and the amount of special anxiety he has previously felt. There is almost always very marked mental distress, and the dread of the disease rather determines the direction of the morbid emotion than causes it. If there has been no apprehension of hydrophobia, as is often the case in children and occasionally in adults (especially of the lower classes), some peculiar delusion may be associated with the emotion, and may seem to be its cause when it is probably its consequence.

In other cases the convulsive symptoms predominate; the spasm induced by attempts to swallow quickly spreads to other muscles besides those immediately concerned, and the tetanoid character may be early assumed. In some instances, again, hysteroid convulsions occur almost from the first, and the respiratory spasm or mere emotion excites the co-ordinated convulsive movement seen in severe hysterical fits. This feature of the spasm may lessen as the disease develops.[*]

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[*] Boerhaave, in his 'Aphorisms,' gave, nearly 200 years ago, a description of the disease so graphic and accurate that it is worth quoting in full from the English translation of Delacoste, published in 1715:

"*Of the dog-madness.*—When a man in perfect health is infected with this poison, after different distances of time he begins to be disordered much after the following manner:—The place pains him where he first receiv'd the injury; then follow wandering uncertain pains in other, but chiefly the nearest parts; a weariness, heaviness, slowness succeeds in the whole system of the muscles; his sleeps are disturb'd, uneasie, full of frights, convulsions, and catching in the tendons; he is continually restless, sighs, looks dejected, and affects to be alone; and it is much after this manner that the disease begins and finishes its first stage. If you bleed

In animals, as we have seen, symptoms of paralysis are much more prominent than they are in man, and sometimes they give a special character to the attack. Examples of the true paralytic form in man are not common, although twenty cases have been collected by Gamaleia.* The cause is usually an extensive bite, but the duration of the symptoms is nearly that of the common form; sensibility is generally preserved, the sphincters involved. The chief morbid changes are in the spinal cord. The onset has been sometimes by sudden paraplegia,† as in the well-known case of the man Goffi. The nature of this case would not have been suspected had not the inoculation test been employed. The symptoms closely resembled those of "acute ascending paralysis."‡ It is important not to mistake, for a peculiar manifestation of the disease, the effects of any potent agent employed in its treatment. The depressing influence of curara on the motor structures has probably been thus mistaken;§ and some other instances of this mysterious form of palsy may have been paralytic rabies.

Pathological Anatomy.—The blood is generally fluid, as in many acute diseases. The throat and pharynx are commonly congested; and so, in many cases, are other organs, especially the spleen and kidneys. The brain and spinal cord often present conspicuous congestion. The most important morbid changes are revealed only by the

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him, then the blood doth not carry the face of any defect. Then are all the foregoing symptoms increased, and there follows besides a prodigious narrowness and pressure about the heart and chest; breathing is difficult, and accompanied with sighs; he frequently shakes all over; his hairs stand an end, and trembles all over at the sight of water, of any sort of liquors, or even upon the sight of transparent things, or reflecting like looking-glasses; he loses his appetite entirely, yet he can swallow anything that is very dry and solid. The touch of any moisture, chiefly with his lips or tongue, creates an incredible anguish, tremors, violent convulsions, and a raving; he vomits tawny, glewish choler or green-like leeks; he grows very hot, feverish, sleepless; is troubled with a priapisme; and he thinks very disorderly of things quite foreign and unusual. Thus doth it go on, and here absolves its second stage. Afterwards doth everything constantly grow worse, and you'll soon see him loll out his rough and dry tongue, gape wide, speak hoarse, have a great drought, grow raving at every attempt to drink, at the sight or touch of any liquor; gather froth in or about his mouth; to endeavour the spitting of the same upon the bystanders, even against his will; to bite and snap at everything within his reach, and that involuntarily, nor yet to be withheld from those attempts; he gnashes his teeth with froth, snarling like a dog; his pulse now and breathing begin to fail, cold sweats break out on all sides, raves in the highest degree, notwithstanding which he is all the while sensible, and is afraid for the bystanders that he shall unwillingly hurt 'em. Hence you may always reckon upon his dying (within the fourth day from the first state of his illness) convulsed with a most terrible anguish upon drawing his breath."

* See Le Gendre, 'L'Ann. méd.,' 1887, No. 40.

† Ricochon, 'Gaz. hebd.,' 1887, No. 10.

‡ See the 'Brit. Med. Journal,' 1886, vol. ii, p. 820.

§ See the comments of Ostermayer in his abstract of an "untypical" case described by Schaffer ('Cent. f. Nervenkr.,' 1891, p. 27).

microscope,* and are found in the central nervous system. They are very variable in their degree, and are sometimes, although seldom, absent. Of nine cases I have examined, morbid changes were distinct in seven. These were indications of vascular disturbance; dilatation of small vessels, accumulations of leucocyte-like corpuscles around them and in the tissues, clots in the small vessels evidently formed during life, and minute hæmorrhages. These changes are met with in various parts, especially in the cortex of the hemispheres, the medulla oblongata, and the spinal cord. They are always most intense in the medulla, between the eminentia teres above and the decussation of the pyramids below, and especially in the neighbourhood of the pneumogastric, hypoglossal, and spinal accessory nuclei. The accumulations of leucocytes about the vessels is a very conspicuous change; they surround the outer wall, and may be so numerous as to fill up the whole space within the lymphatic sheath (Fig. 168); they may extend along the vessel for a considerable distance, and even pass into the adjacent tissue (Fig. 169). The

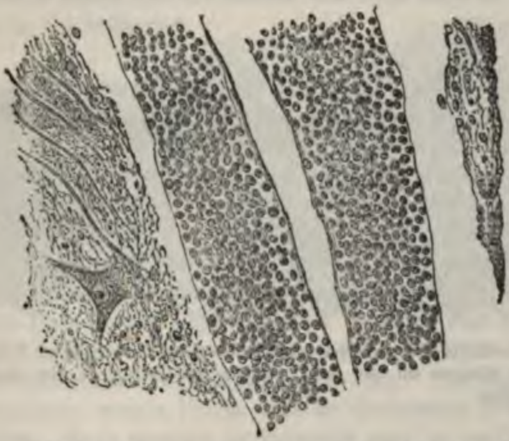

Fig. 168.—Hydrophobia; perivascular sheath distended with leucocytes; from the hypoglossal nucleus.

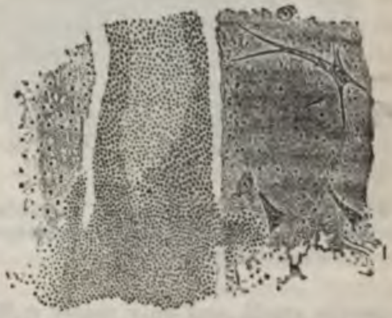

Fig. 169.—Hypoglossal nucleus; leucocytes around a vessel, and extending into the adjacent tissue.

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* They have been investigated and described by Coats, Clifford Allbutt, Benedikt, myself, and others. The figures here given are from a paper of my own, published in the 'Path. Trans.,' vol. xxviii.

nuclei contain a much larger number of such corpuscles than normal and in places they may be aggregated and form dense masses (Fig. 170), which, since the corpuscles may be regarded as identical with pus-cells, are in fact "miliary abscesses." These collections may sometimes also be seen outside the grey matter, scattered at random, as it were, especially about the root-fibres of the nerves (Fig. 170). The thrombi within the vessels fill their cavity, and may present curved lines as if they had been exposed to the pressure of the blood. At the seat of these clots the walls of the vessel may be distinctly thickened. The nerve-cells sometimes appear swollen and slightly more granular than under normal circumstances, but their alterations are trifling in the hardened specimens in which they are commonly examined. The hæmorrhages are usually small, and only rarely sufficiently large to be visible to the naked eye. In the spinal cord the alterations are usually much slighter, and are confined to encrustation of the vessels with leucocytes and to an increase in the number within the grey matter; in this the changes are usually greater than in the white columns, although they involve these in severe forms. They may, indeed, be so intense as to constitute a condition practically of acute myelitis. Even when a case has run so rapid a course as to be fatal in three days such myelitic changes may be conspicuous.*

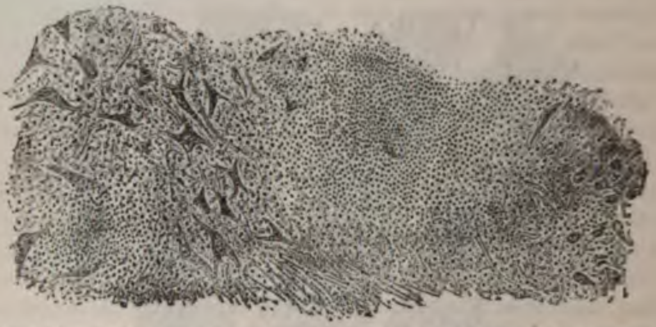

FIG. 170.—Cells of the hypoglossal nucleus on the right, of the pneumogastric nucleus on the left. Dense infiltration of the tissue with leucocytes.

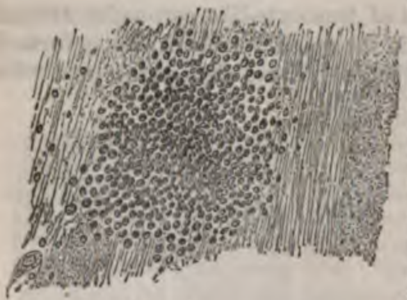

FIG. 171.—Accumulation of corpuscles ("miliary abscess") in the fibres of origin of the hypoglossal nerve.

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* Schaffer, 'A. f. Psych.,' xix.

The "miliary abscesses" are very seldom seen in the cord. On the other hand, the central nervous system may present no other alterations than are common after death from asphyxia, and this even in an animal inoculated with rabies,* as well as in man. Outside the nervous system leucocytal infiltration has been seen in the salivary glands and in the kidneys.†

Pathology.—It may be well, in considering the pathology of the disease, to distinguish the facts that are certain, or possess a probability almost equivalent to certainty, from the mere hypotheses that afford at present the best explanation of the phenomena observed.

(1) It is certain that the disease is the result of a virus, not volatile, and transmissible only by inoculation. That it is a micro-organism has not yet been demonstrated, but does not admit of doubt. The virus seems to increase within the body, and so causes the disease; Pasteur has shown that it develops best and chiefly in the central nervous system, where inoculation (in rabbits) never fails, as it sometimes does beneath the skin. The virus certainly varies in intensity, within limits, and so (inversely) does the period of incubation. Such increase in intensity may be produced by the successive inoculation of a series of animals with full doses. The position of the anatomical changes, coupled with the character of the symptoms, shows that the virus acts most intensely on certain parts of the medulla oblongata, next on the spinal cord, next on the cortex of the brain. But the variable degree of the changes, and their occasional absence, prove that they are secondary effects of the disturbance of the function of the nerve-elements by the virus, an influence known in other diseases to have vascular disturbance as its consequence, although the latter, if intense, may have its own effects.

(2) We must regard as hypothetical the explanations that have been given of the variable and often long interval between the bite and the symptoms. The minimum interval may fairly be regarded as a true "incubation" period, and slight variations in it may be referred to the intensity of the virus; but for the much longer period that is so common we are still without any better explanation than the old one, that the virus is lodged for a time in the vicinity of the wound, and at last dislodged by some accidental influence, such as gymnastic exertion, to which this effect was ascribed in one case. But two, if not three, questions need consideration in connection with this problem. We must assume that both organisms and germs are introduced; that the symptoms, since they never occur at once, are not due to the former, which perish, probably often leaving other germs; and that the shorter "incubation" period is the time needed for the development of a sufficient number of organisms from the germs, perhaps within the nerve-centres. The localisation of the germs near the wound, but beyond the reach of tardy cauterisation, is

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* Rovighi, 'Riv. Clin.,' 1886.

† Coats, Hale White, &c.

easier to conceive than local persistence of developed organisms. When malaise, &c., precede the onset for some weeks, it is possible that the organisms first developed are too few to produce the disease, which only results from the maturity of a second, far more numerous "brood." Many forms of organised virus seem only to break down the resistance of normal function where they possess a certain degree of intensity.

It has been extensively held (by Pasteur and others) that the path of the organisms from the wound to the nerve-centres is by the nerves, and by this slower transit the long interval is explained; but the theory is improbable, and the interval is too variable to be thus explained. Such a transit would certainly entail indications of intense neuritis, invariable and preceding the onset. All facts suggest that the organisms introduced soon die, and that germs introduced or generated are effective by their development in the nerve-centres. There is no difficulty in conceiving that they reach these by the blood. The entrance of a few germs into the peripheral nerves, and development there at the same time as (or a little before) the chief development takes place in the central organs, is a possible explanation of the local nerve-pains experienced in some cases.

From the facts of the disease, and from what we have learned of analogous maladies, we may assume that the symptoms are due to the direct action of the virus on certain parts of the nervous system. The active agent, in some allied diseases (as tetanus), is a chemical product of the organisms. No facts have yet been ascertained to suggest that this is true of rabies, although it cannot be considered unlikely. There is reason to believe that the organisms, by their growth, produce a material incompatible with their continued existence in the system. It is on the latter theory that Pasteur bases the explanation of his prophylactic treatment, and we shall have to return to it in considering the latter.

The way in which the virus acts is suggested by the character of the symptoms. Its primary effect on the nerve-centres seems to be to increase their activity, and especially to augment the reflex excitability of the medulla oblongata and of the spinal cord, while corresponding disturbance of the cortex causes the delirium, &c.; the effect on the medulla oblongata increases the excitability of the respiratory centre; and, as the extraordinary muscles of inspiration (not the diaphragm) are chiefly excited by reflex influences, the inspiratory efforts are of the "costo-superior" type. The centre for these movements is adjacent to that for deglutition, and the region of the medulla in which the changes are most intense is that in which the glosso-pharyngeal, pneumogastric, and hypoglossal nerves are situated. The effect of the poison seems to be chiefly on these centres, just as other organic poisons have specific actions on other parts of the nervous system. The period of excitement is often succeeded by the opposite condition, shown by the paraplegic weakness and coma that may supervene. But another agency may be concerned in the abolition of function, viz. the vascular changes that are revealed by the microscope, and which, as we have just seen, must be regarded as secondary results of the functional over-action.* These are somewhat random in distribution; the exact points at which they occur are apparently influenced by conditions which are, so far as the disease is concerned, accidental, the specific poison only determining their general situation. When they do occur, however, the leucocytal infiltration may be so great as to break up the tissue, and even to constitute a minute point of suppuration; and if the point thus affected is important, grave paralytic consequences may ensue in both the medulla oblongata and the spinal cord. Further, it is probable also that the cortical excitement acts upon the centres of the medulla oblongata and increases their disturbance. We know how much the functions of the pneumogastric are under mental influence; the sigh, and vomiting from disgust, are examples. On the other hand, it is possible that the disturbance of the medulla may determine the direction of the cerebral derangement. We may often trace, in the symptoms, evidence of this peculiar mutual interaction of the bulbar and cortical disturbance.

It is important to note that some of the symptoms of hydrophobia not only resemble, but are identical with those of hysteria. The convulsion may be distinctly of hysteroid character, and the frenzied horror at the sight of water has a close parallel in the maniacal stage of many hysterical attacks. In connection with the barking and biting of the hydrophobic patient, it should be remembered that both actions may occur during the attacks of pure hysteria; the biting is especially common.† There is nothing in itself extraordinary in hysteroid disturbance forming part of the functional disturbance of the brain that results from a different cause, but the therio-mimicry of hysteria is a mysterious phenomenon, the significance of which is doubtless great, though at present beyond our observation. It is not lessened by the prominence of the condition in a malady due to a virus exclusively animal, especially since it cannot be altogether explained by the assumption of mental preoccupation.

The special paralytic form seems to be due to the action of the virus being primarily and specially on the spinal cord, and being from the first depressant in its influence. We cannot at present explain the last fact, but some light is thrown on the first by the fact‡ that injection of the virus into the veins causes the paralytic form, while subdural inoculation always gives rise to the ordinary variety with initial excitement. Through the blood the virus can evidently have free access to the spinal cord. But the difference in its influence in the two cases suggests some difference in the virus, and in the character of its action on the nerve-elements, to which we have at present no clue.

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* Hence it is incorrect to speak of inflammation of the medulla as the effect of the poison and the cause of the symptoms, and to make a comparison, as has been done, between the condition of the medulla in hydrophobia and that of Peyer's patches in typhoid fever.

† See the description of this disease.

‡ Pasteur, confirmed by Szpilmann.

Diagnosis.—The symptom of greatest value, as indicative of the nature of the affection, is unquestionably the peculiar respiratory spasm excited by attempts to swallow liquids. The history of a bite usually gives significance to definite symptoms, but occasionally misleads when these are equivocal.

In certain acute diseases of the throat and chest, sudden dysphagia has given rise to a suspicion of hydrophobia, chiefly when the dysphagia is not due to pain, but is reflex, a throat spasm. Such cases are, however, very rare, and the history and other symptoms are distinctive if ordinary care is used. It is more likely that these affections should be mistaken for hydrophobia than that the latter disease should be overlooked. The chief diagnostic difficulty is in connection with nervous disorders. Cases in which the mental disturbance preponderates may be mistaken for simple insanity, but the associated spasm, significant even if slight, and the onset after a suspicious bite, generally prevent error. If there is a form in which spasm is absent, the distinction from simple insanity may be only possible by the extremely rapid course of the affection. No patient ever passed from mental health to a state of imminent danger in two or three days in consequence of simple insanity. The opposite mistake is far more common; a previous bite leads to an erroneous diagnosis of rabies.

Organic brain diseases—meningeal hæmorrhage, for instance—occurring in a person who had been bitten by a dog, have been mistaken for hydrophobia. One case is recorded in which the mistake was only discovered at the post-mortem examination. The character of the convulsions and delirium, and the absence of respiratory spasm, should prevent the error. From tetanus, hydrophobia is distinguished by the late period after the bite at which the symptoms develop. A difficulty can scarcely arise (except in the case of idiopathic tetanus) unless the patient has received some recent wound of another nature. The respiratory spasm and aversion to liquids in the one case, and the early trismus in the other, almost always suffice for the diagnosis. If the symptoms of hydrophobia are present, the association of general tetanic spasm with the respiratory disturbance is quite compatible with the hydrophobic nature of the case.

Symptoms bearing a superficial resemblance to hydrophobia sometimes occur in those who have been in great dread of the disease, in consequence of a bite from an animal possibly or certainly rabid. The knowledge that difficulty in swallowing is a symptom of the disease, determines the occurrence of spasm in the throat on deglutition, perhaps analogous to the "globus hystericus." The patient's fears are intensified as the spasm increases in degree, and a dread of water may come on. Hysterical convulsion may be added, and the group of symptoms produced may deceive even the medical attendant. In this "lyssophobia," or "pseudo-hydrophobia," as it has been termed, the most important diagnostic element is the absence of all true respiratory spasm; there is not the peculiar "catch in the breath" that characterises the genuine disease. There has always been much preceding mental alarm, but too much weight must not be placed on this, because great anxiety of necessity often precedes true hydrophobia.* The symptoms of the spurious form commonly soon subside if the mind of the patient can be tranquillised, or if the patient has recourse to some method of treatment in which he can be induced to place confidence. An instance of this is afforded by the case of Dr. Buisson, who after receiving on a scar some saliva from a patient who was suffering from hydrophobia (real, or more probably supposed) thought himself suffering from the disease. He took a vapour-bath and was well. He afterwards treated forty cases of the same character successfully in this manner! But there are other cases, regarded as spurious, in which the mental disturbance is intense, spasm distinct, and the patient passes into a state of exhaustion and dies in a few days. It is probable that most of these cases are genuine, although they have been published as spurious.†

I have more than once known a first attack of hysteroid convulsion, in a lad who had been bitten by a dog some time before, to be mistaken for hydrophobia on account of the barking and biting and general convulsion. The absence of the respiratory spasm, and the intermitting character of the disturbance, should prevent error, which, indeed, can only occur if the characteristics of severe hysteria are not known.

Prognosis.—Attacks of hydrophobia differ in their intensity and in the rapidity of their course, but at present all we can say is that in any given case, however mild, only one issue can be looked for. It is to be hoped that the future may render the prognosis less grave; although we seem as far as ever from an actual cure for the developed disease, we may still echo the hope, expressed by Boerhaave nearly two hundred years ago, "Nor ought we yet to despair of finding out, some time or other, a peculiar antidote for this poison, seeing we have succeeded in poisons thought formerly as destructive."

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* There has more often been a tendency to regard the genuine disease as imaginary than to mistake the spurious for the genuine. This tendency is especially marked among critics who have not seen the case, who do not scruple to cast doubts on the nature of even fatal cases. It is not certain that death has ever occurred from mere "lyssophobia."

† The diagnosis of the spurious from the genuine form of the disease is thus a matter on which there is very wide difference of opinion, which involves also the diagnostic indications. This difference will probably be lessened considerably now that we have an apparently crucial test in the results of inoculation. Two fatal cases described as "pseudo-hydrophobia" have been published by Broadbent ('Clin. Soc. Trans.,' Feb. 23rd, 1883), the spurious character of which appears to me to be open to question. In one case it is true the bite was received five years before, but this ought not to be allowed to *determine* the diagnosis (see p. 926).

TREATMENT.—*Preventive.*—The prevention of a disease is important in proportion as its treatment is ineffectual; and the fatality of hydrophobia renders its prophylaxis of paramount urgency. The prevention of hydrophobia is the prevention of rabies, and this could be readily effected in Great Britain on account of our insular position. The enforced muzzling of all dogs for a period of one year would almost certainly stamp out the disease.* It cannot be doubted, also, that the number of dogs is vastly in excess of any real need of the community. Most cases of hydrophobia are due to animals kept for pleasure, not for use, and often ill-kept. More might also be done to make those who keep dogs familiar with the early symptoms of rabies, and to render it a criminal offence to permit a dog that presents such symptoms to be at large.

Until this wise and humane measure, the universal use of the muzzle, is adopted, the prevention of hydrophobia centres in the method devised by Pasteur, and in the cauterisation of the bite. Pasteur's system of prevention depends on the fact that by drying the spinal cord of rabbits that have been rendered rabid by inoculation, the virus becomes less active; the subcutaneous injection of an emulsion of such a cord (dried for fourteen days) causes no definite symptoms of the disease, nor do subsequent inoculations or injections made with cords dried for a less time. After such inoculations an animal is "protected," and does not suffer from rabies if afterwards bitten by a rabid animal, or even if inoculated with the virus in its most active state.† These conclusions have been confirmed by Horsley,‡ in a series of experiments on dogs, which seem to be free from any source of fallacy. In these experiments the preventive treatment was carried out before the infection. Pasteur further believes that such a course of injections is effective in preventing the occurrence of the disease in a person who has been already inoculated with rabies, and he has treated thus many thousands of bitten persons. The efficacy of the treatment has been the subject of much discussion, and so also has its safety. Two facts only are certain. First, the danger that rabies may be communicated by the treatment is very small. There is, indeed, one case in which this result is rendered probable by the very unusual character of the symptom, the case of the man Goffi, who was bitten by a rabid cat, and died of paralytic rabies in St. Thomas's Hospital (see p. 930).[*] But this case only brings into relief the immunity with which the course of treatment has been pursued in an enormous number of cases.[†] The other fact is that the treatment is not invariably successful. In several instances, rabies has developed at about the same time, after the original bite, that it might be expected to develop had the treatment not been adopted, and its severity has not apparently been modified. The efficacy of the treatment has therefore to be judged from statistics, and the difficulty of drawing a conclusion is great, and is increased by the fact that, of a given number of persons bitten by a mad dog, only a proportion, sometimes small, suffer afterwards from the disease (see p. 925). But the proportion who have suffered after undergoing the Pasteurian treatment is certainly far smaller than it would be in a like number of persons who had not been thus treated. Up to the end of 1886, 2682 persons had been inoculated, and the subsequent mortality from rabies among them was between 1 and 1·2 per cent. Even allowing largely for doubtful bites it is estimated that not less than 5 per cent. of such a series would have died without the treatment.[‡] We cannot feel sure that in all cases the animal that inflicted the bite was actually rabid, and hence a new source of uncertainty is introduced, the influence of which cannot be accurately assessed. The Committee on Hydrophobia investigated the facts of ninety unselected cases treated by Pasteur, and in two thirds of these there was evidence which satisfied the committee that the dog was rabid. In no less than twenty-four the bites were on naked parts, and the wounds were not cauterised or treated in any way likely to prevent the action of the virus. Every one of these ninety patients had continued well, although all had been bitten more than six months, and most of them more than a year before. In such a number of cases of this character it was calculated by the committee, on the lowest estimate, that not less than eight would have suffered from hydrophobia. The conclusion from the facts at present available seems, therefore, to be that the treatment is of great value, although it does not secure absolute safety, and that the method is not attended by more than an extremely small risk of the production of rabies.

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* That such a measure is not adopted is a national disgrace, which is accentuated by the fact that the Government derives part of its revenue from a tax upon dogs. The opposition to the use of the muzzle is one of the strangest developments of morbid sentiment. There are apparently thousands of well-meaning people who would prefer that hundreds of dogs should perish every year of a painful malady, that many human lives should be annually lost, and scores of persons should be subjected for months to acute mental agony—rather than that dogs should be made to wear an apparatus which causes them a trifling annoyance for a few days. This perverted sentiment ought to be met with universal abhorrence, as a disgrace to humanity.

† Pasteur puts forward the theory, already refered to—which has however, nothing to do with the accuracy of his facts—that the virus, a micro-organism, produces, in its development, some substance by which its growth is checked, as is the growth of yeast by the alcohol produced. He thinks that the spinal cord contains both the virus and the antidotal product, and that the antidote is much less influenced by the process of drying than is the virus.

‡ 'Report of Committee of Inquiry into M. Pasteur's Treatment of Hydrophobia,' 1887.

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[*] The result has been ascribed to the circumstance that the man was almost constantly drunk while undergoing the treatment.

[†] It has been suggested that some of the deaths from the ordinary form of rabies, after the treatment, were due to the inoculations, but the case referred to renders this doubtful. It suggests that the inoculation with the rabbit's cord would produce the paralytic, and not the ordinary form.

[‡] 'Report of Hydrophobia Committee,' p. 3. Some further facts are very important. Of 233 persons bitten by animals in which rabies was certain (either by inoculation from the spinal cord or from the occurrence of rabies in other animals or persons bitten) only 4 died, while without inoculation 40 would probably have died. The mortality among persons bitten by rabid wolves is exceedingly high; the bites are generally severe, and prompt cauterisation is impossible: 48 persons so bitten were treated; a mortality of 30 would have been almost certain, but only 9 died, and in 3 of these the symptoms commenced while under treatment. One very striking group of cases is that of six children bitten at Bradford, January 24th, 1886, by a dog proved to be rabid by experimental inoculation, and by the fact that another person who had been bitten by the same dog died of hydrophobia on March 4th of the same year. The six children who were treated were perfectly well at the date of the report, eighteen months after the bites. A more crucial test could hardly be devised. Equally strong is the instance of three children bitten at Shipley, Yorkshire, in March, 1886, and treated by Pasteur, all of whom remained well a year later, while another person bitten by the same dog, and not treated, died from hydrophobia seven weeks after the bite.

Treatment of the Bite.—The poison is deposited in a wound, and if it can be destroyed before it passes into the blood,[*] the disease will be prevented. The measures to be adopted for this purpose belong to the province of surgery, but may be briefly mentioned. If the wound is on a limb, the circulation should, if possible, be arrested by a ligature around the limb, applied immediately. Free bleeding should be encouraged and the wound should be well washed. These measures are most important, because it is not often that the cautery can be immediately applied. The use of these measures in the case of bites of every kind ought to be part of the education of children. The wound may be sucked, if there is no abrasion in the mouth, which should be washed out each time. Absorption of poison through a mucous membrane (to judge from experiments on the conjunctiva) occupies several minutes. As soon as possible, the wound should be thoroughly cauterised by the actual cautery, nitric acid, or nitrate of silver. Whenever practicable the bitten part should be excised.

The treatment of the developed disease has to be directed chiefly to the diminution of the suffering of the patient. At the same time recovery cannot be said to be impossible. It is certain that cases have recovered in the past, and it seems therefore certain that cases will recover in the future. However slender may be the hope of doing more than lessen suffering, the patient should not be given over into the hands of death, even indirectly by efforts exclusively directed to the euthanasia, still less ostensibly, as by the feather beds of old.[†]

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[*] This is the common theory of cauterisation, but is hardly consistent with the assumed localisation of the poison in the wound during the period of incubation. It is, however, certain that cauterisation is of little value unless it is almost immediate. Promptness is of more importance than the agent employed. A smoker's "vesuvian" would probably be quite effectual if instantly used.

[†] It is curious to note how the name of the disease preserves the fact that the horror of water was formerly regarded not only as a characteristic of the disease,

The pathological lesions show that the functional nervous excitement has organic consequences, and it is important, in order to lessen these, and to maintain the strength of the patient, that whatever excites the paroxysmal disturbance of the nervous system should be carefully avoided. A dimly lighted, quiet room should be secured, and only the necessary attendants should be admitted into it. Every sight or sound likely to disturb should be avoided, and it would probably be wise, if the spasmodic dysphagia is great, to feed only by peptonised enemata.

The drugs that have been employed in the treatment of the disease are innumerable, and embrace (besides most known sedatives) a number of alleged specifics, all of which have been abundantly proved to be useless. The treatment employed in the few cases in which recovery has taken place, has generally been found powerless in other instances.

One case is said to have recovered under the influence of mercury, but this has been tried since in numberless instances without result. Curara, however, has been credited with three cures (Offenburg, Polli, and Watson), of which one case, that of Offenburg, seems undoubtedly genuine. The drug was first recommended half a century ago by an Englishman, Sewell,* but tried in small doses it failed. It arrests the hydrophobic spasms by paralysing the motor nerves, and this may render artificial respiration necessary. There seems to be a remarkable tolerance of curara in this disorder. The dose commonly employed has been from $\frac{1}{16}$ to $\frac{1}{2}$ of a grain repeated every quarter or half hour until there is general muscular paralysis, and renewed as this effect passes off. Polli administered in all three grains in five and a half hours to a child twelve years old. Even grain doses have failed to cause paralysis.† One case, probably genuine, has been recorded, in which recovery occurred under the use of Calabar bean and hypodermic injections of morphia (Nicholls). Bromide of potassium has very little influence; if given at all it should be in large doses, one or two drachms. Chloral gives some relief, and, in a case recorded by Sansom, life was prolonged for ten days under its use. It is probable that the combination of chloral and morphia, which has a special

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but as the predominant element in its pathology, which afforded the chief therapeutical indication. "In the intervals" (of venesection, says Boerhaave) "he must be blinded and thrown into a cold pond, or be made wet with the continual throwing of water upon him, till he doth not seem any more to be afraid of water, or but little," a final reservation that was at least prudent.

* And also by Waterton, the naturalist, who brought to England a supply of the drug (see 'Dolan's Hydrophobia,' 2nd ed., p. 170, and Waterton's 'Travels').

† But it is possible that in some cases the agent has not been very active. Thus, in a case recorded by Bristowe, in which as much as $1\frac{1}{2}$ grains were injected at a time, and 8 grains in twenty-four hours produced very little effect, the curara "had been in stock some time, and there was reason to believe that it had deteriorated in quality" ('Brit. Med. Journ.,' April 28th, 1885). The activity of the specimer should always be tested.

influence on the respiratory centre, deserves further trial, and on the whole constitutes, as far as is at present known, the wisest treatment.

Tracheotomy was suggested by Marshall Hall as a means of averting death during an asphyxiating attack of spasm, but it is probably powerless. The spasm involves the chest wall, and, according to Pitt,* the glottis is widely open during the attack.

Of the various remedies that have been held in popular esteem or vaunted as private specifics, little need be said. They furnish a curious chapter in the history of charlatanism and credulity. "Hitherto," we may still say with Boerhaave, "we have not met with any one which deserves credit enough to be entrusted with the life of people thus miserably afflicted, for no one is known whereof the experiments be certain, but they owe their birth either to speculation, or they have been copied from others and taken upon trust." The list of reputed remedies that he gives has received a long addition since his time. Persons who fancy that they are suffering from the disease are cured by these measures, and their recommendations are repeated with confidence by those whose readiness to express an opinion is inversely proportioned to their capacity for forming a judgment. Foremost among these measures is the vapour-bath of Buisson, the origin of which has been already mentioned (p. 937), and which every few years makes its reappearance in the public papers.

The saliva of persons suffering from hydrophobia has been proved to be capable of communicating the disease to animals. Hence the attendants should be cautioned to have no uncovered abrasions on the hands, and to wash from the eyes and face any saliva that may have been spit on them. If they are bitten by the patient, the wound should be treated as if it had been inflicted by a rabid animal. These precautions remove all danger, and any anxiety that is felt may be removed by the assurance that, of the thousands of persons who have attended on patients with hydrophobia, no authentic instance has been recorded in which the malady was thus contracted.† Nor has it ever been known to have been acquired through making a post-mortem examination on the bodies of persons who have died from the disease.

METALLIC POISONING.

Many metals, when taken into the body, cause chronic disturbance of the nervous system, in addition to the acute symptoms that are produced by a considerable dose of the poison. The latter are not considered here, since they belong to the province of toxicology. The more chronic disturbance generally results from the often repeated entrance of small quantities of the metal, and seldom succeeds a single large dose. The chief metals that influence the nervous system are lead, arsenic, silver, and mercury.

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* G. N. Pitt, 'Med. Times,' June 20th, 1885.

† Indeed, even the danger of bites seems small. A child, whose malady was verified by the inoculation test, bit two persons whose wounds were uncauterised, and at the end of six months they were still well ('Riv. Clin. di Bologna,' Aug., 1886).

LEAD-POISONING.

ETIOLOGY.—The occurrence of symptoms of lead-poisoning seems to be, to some extent, determined by individual peculiarities. Of a given number of persons, all of whom have been exposed to the same influence, and exhibit indications of the presence of lead in the system, some will suffer considerably, some slightly, others not at all. Little is known of the conditions which thus influence the result, but females seem more prone to suffer than males, the weakly than the strong, and those who inherit a tendency to gout, especially if the affection, thus inherited, was due to the same cause. Disturbances of the nervous system are much increased by intemperance; the influence of alcohol in evoking cerebral symptoms is not only observed in man, but has been noted in animals.

The duration of exposure, before symptoms occur, varies, and is chiefly determined by the amount of lead daily absorbed. When this is large, severe symptoms are often induced in a few weeks. Some of the most acute forms occur in young persons whose exposure has been brief. It is probable that, when the daily dose of the poison is small, a certain amount of tolerance is sometimes established, which may be ultimately overcome in consequence of the accumulation of lead in the system, or in consequence of some impairment of the general health.*

The sources of lead-poisoning are very numerous, but may be divided into two groups, the *industrial* and the *accidental*. Of the former, the most potent are lead-works, especially those in which white-lead is prepared, but more frequent are the various industries in which prepared lead is used. Painters, plumbers, type-founders, compositors, and those who glaze pottery with lead, are the most frequent sufferers. Glass grinders may also suffer, lead being a constituent of most kinds of glass. Various other occupations involve liability to poisoning, often unsuspected. Licking gummed paper, tinted with colouring matter containing lead, and the process of preparing cards with a lead glaze, have been effective. The *accidental* sources are extremely numerous; the most frequent is the contamination of drinking-water by leaden pipes, or by lead-lined cisterns.†

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* On these and most other facts relating to the subject, numerous original observations, clinical and experimental, will be found in 'Lead Poisoning,' by T. Oliver, M.D., Edinburgh, 1891.

† Various chemical peculiarities of water will make it take up lead more readily. For particulars the reader is referred to works on hygiene. A curious instance is related by Thorne in which lead-poisoning at Sheffield was due to the presence in the water of a peculiar acid derived from the soil ('Practitioner,' Dec., 1886, p. 465). This danger from water was known two thousand years ago.

Acid fruits, cooked in glazed earthenware vessels, have become charged with lead; shot left in wine bottles, after cleaning, has been dissolved by the wine or "mineral water;" snuff may be contaminated in consequence of being packed in lead-paper. Lead enters into the composition of some hair dyes and cosmetics, and cases of lead-poisoning have occurred from their use. Infants at the breast have been poisoned by a lead ointment applied to excoriated nipples. Lastly, symptoms of chronic poisoning have occurred from the continued medicinal administration of lead.

The most frequent path by which lead enters the system is the alimentary canal, not only by the accidental contamination of articles of food, but also through uncleanliness. Workers with lead often neglect to remove all trace of lead from their hands before taking food, and thus become poisoned. This is shown by the influence of enforced cleanliness, which lessens the amount of lead-poisoning among such artisans. The saliva dissolves but little of the carbonate of lead (the form in which the metal is most frequently received). The hydrochloric acid of the gastric juice transforms it into soluble chloride, and the bile also dissolves it, but in each case products of digestion hinder the process, insoluble albuminates being formed, and, as a rule, most of the lead passes away by the bowel (Oliver). When lead enters through the skin, as in poisoning from the use of cosmetics and hair dyes, it may be dissolved by some of the organic acids secreted by the cutaneous glands, or get through the skin in consequence of the friction employed in rubbing hair dyes into the roots of the hair. Lead-poisoning has been produced in children by the continued use of Goulard's lotion.[*] Lead may be thus absorbed by those who work with the hands immersed in preparation of the metal. It may certainly enter by the mucous membrane of the nose (in the case of poisoning by contaminated snuff), and probably also by that of the other air-passages; inhaled particles are dissolved by the alkaline secretion, or enter the blood and are transformed into soluble bicarbonate by the carbonic acid contained in it. No doubt, however, some lead, entering by the air, is carried by the buccal secretions to the stomach.

It is commonly believed that, however lead enters the system, it acts on the tissues through the blood, permeating the whole body and influencing chiefly certain structures. Unquestionably, many of the symptoms must be thus produced.[†] But lead has been said also to have a local influence on the part where it comes in contact with the skin. This opinion, although based on some curious facts, has not received general confirmation.[‡]

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[*] It has been readily produced in animals by an ointment of oleate of lead and lanoline (Oliver).

[†] Although some are ascribed to indirect effects, due to the deranged action of the liver and kidneys (Oliver).

[‡] The facts that suggest a local influence are mentioned at a later page.

The lead which has entered the system collects in the various tissues and organs, but not in the same degree in all. In dogs, to which lead is given in food, the organs have been found to contain the metal in the following order:—bones, kidneys, liver, spinal cord, brain, muscles, intestine; elsewhere there is only a trace (Heubel). It is not certain that this order is always the same in man. In one case, for instance, much more lead was found in the brain than in the liver, and none in the spinal cord (Troisier and Lagrange); while in another case the liver contained much and the brain none.* In even acute cases lead has been sometimes absent in the brain.†

Lead is excreted by the liver; also, it is said, by the intestinal mucous membrane, but in man chiefly by the kidneys. The urine always contains lead, when this is present in the blood; the quantity of urine is at first increased and subsequently lessened; at the last, there may even be anuria, and with it intense disturbance. As a rule, the amount of uric acid excreted bears an inverse relation to the amount of lead. When injected under the skin of an animal, almost all has been said to pass away by the bile,‡ but this does not seem to be the case in man. Once combined in the organs, it is very slowly eliminated, and its presence may be detected years after it has ceased to be taken in. It probably exists in them in some combination with albumen, which may not be always the same. But the amount fixed in the tissues is very small compared with that which is eliminated from the blood, and the quantity that enters the blood is small compared with that which enters the stomach.

SYMPTOMS.—Disturbance of the general nutrition usually precedes other symptoms, although it may escape observation. There is anæmia, often considerable, depending on an actual diminution in the number of blood-corpuscles. The hæmoglobin is reduced in proportion to the corpuscles, and the anæmia of lead thus differs from that of chlorosis, in which the reduction is much greater in the hæmoglobin than in the corpuscles. Lead-anæmia resembles in this respect pernicious anæmia, and the resemblance is not merely superficial. The condition (which is readily produced in animals) is probably due to accumulation of lead in the bones, the medulla of which is known to be an important blood-making tissue. Atrophy and degeneration of the marrow have actually been found (Raimondi). The general nutrition and muscular strength are much impaired. As a rule the temperature is normal, but now and then there is slight continuous pyrexia; in one case under my observation the temperature was constantly raised to 99° or 100°. When acute nervous symptoms occur, elevation of temperature is common. In a few cases endocarditis has been observed; degeneration of the cardiac valves may occur in chronic cases, as part of the slow changes in the vascular system.

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* Pye-Smith, in Fagge's 'Manual,' ii, 163.

† Comby, Oliver.

‡ Lehmann, 'Diss. Berl.,' 1882; Prevost and Binet, 'Rev. Suisse,' 1889. Lead may also be eliminated by the skin, as is proved by a case of poisoning by red-lead, recorded by Oliver, in which the linen in contact with the skin was reddened by the perspiration.

The subjects of lead-poisoning often suffer from gout when they have reached middle or later life*—an important fact, alike from a diagnostic and pathological point of view. It must be associated with the lessened elimination of uric acid, although the nature of the association is not certain, and will be again referred to. The kidneys soon become diseased, first from a form of parenchymatous nephritis, which tends to become interstitial, and at last, as in ordinary gout, the kidneys are often indurated and granular. Apart from actual kidney disease, albumen may be present in the urine at times, as, for instance, during attacks of colic (Seguin). The effect on the kidneys is very important, since it leads to cardiac and arterial disease, and thus to cerebral lesions. By this mechanism, more frequently than by any other, lead-poisoning causes death. Kidney disease may slowly follow lead-poisoning which has ceased. A gentleman suffered from wrist-drop due to lead-poisoning from drinking-water; his urine was then normal. There was no further ingestion of lead, and he recovered perfectly, but six years later presented all the signs of organic kidney disease.

A characteristic symptom is a narrow, bluish-black line on the gums, close to the teeth, distinguished by its narrowness and tint from the more common zone of purplish congestion. The lead line forms where the gum is not in perfect connection with the tooth, and it is really the edge of a layer of deposit beneath the inner, detached surface of the gum (Cras). It may be first seen on the projections of the gum between the teeth. It consists of sulphide of lead, deposited chiefly outside loops of blood-vessels, and hence, when viewed with a lens, it often has a punctate appearance. The sulphur comes from albuminous matters which accumulate between the gum and the tooth. If the gums are not detached from the teeth, and these are kept scrupulously clean, the lead line may be visible only between the teeth, or it may even be absent entirely. A punctate deposit of sulphide of lead may form in the mucous membrane of the lip, upper or lower (as Garrod first pointed out). It is only seen when there is an accumulation of tartar on the teeth with which the stained lip has been in contact. A low form of stomatitis has been occasionally observed, and sometimes ulceration occurs on the inner surface of the cheek and elsewhere.

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* This was originally pointed out by Garrod. "Saturnine gout" is invariable in those whose parents were gouty, and so frequent that most sufferers from chronic lead-poisoning are distinctly gouty. This is at least the case in the south of England. It is said not to be so in the north, where gout is both uncommon and a rare effect of lead-poisoning, unless the latter is reinforced by an inherited tendency to gout (Oliver).

Severe abdominal pain, "lead colic," is another frequent symptom. It is a paroxysmal pain, felt chiefly in the region of the umbilicus, sometimes over a more extensive area, and accompanied with obstinate constipation. During the paroxysms there is retraction of the abdominal wall, sometimes vomiting, and usually retardation and increased tension of the pulse. The constant constipation proves that the pain is not (as has been sometimes maintained) in the abdominal wall. It is apparently due to a true "colic," the result of a spasmodic contraction of the bowel, probably the colon—a contraction which is tetanic and not peristaltic, and therefore arrests, instead of promoting, the movement of the intestinal contents. The constipation may be facilitated by a diminution in the secretion from the mucous membrane, degenerative changes in which have been found. Such tetanic contraction has also been produced in animals. Its mechanism is uncertain. It has been ascribed to the influence of lead on the sympathetic ganglia, especially the mesenteric and cœliac plexuses, on the nervous ganglia within the intestinal wall, on the muscular fibres of the bowel itself, and it has also been supposed to be connected with the accumulation of sulphide of lead in the mucous membrane, sometimes visible after death.* At present there is no evidence to show which of these theories is correct, and it is therefore uncertain whether lead colic should or should not be classed among the nervous effects of lead-poisoning. The retardation of the heart's action, and the increased tension of the pulse, associated with the colic, are doubtless, in part at least, reflex effects of the strong afferent impression. Severe paroxysms of epigastric pain have also occurred, associated with inaction of the intercostal muscles, apparently inhibitory.

Nervous Symptoms.—The definite nervous symptoms produced by lead-poisoning are of five chief classes: (1) Pains in various situations. (2) Muscular paralyses with wasting, acute or chronic. (3) Paraplegic symptoms. (4) Cerebral disturbance, acute and chronic, transient or increasing; convulsions, delirium, coma, amaurosis, hemianæsthesia. (5) Affections of certain cranial nerves.

1. *Pains in the limbs* are very common, and may be referred to the muscles or the joints, or may be vague in seat. They are usually dull, aching, "rheumatic" pains, but sometimes sharper and neuralgic in character. The joint pains have been called "saturnine arthralgias," but, apart from gout, articular pain is not usually prominent. There is often tenderness of the muscles, and occasionally tingling of the extremities, rarely areas of anæsthesia. I have seen, for

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* Maier ('Virch. Arch.,' Bd. xc) found degeneration of the nervous ganglia in the wall of the intestine, and in the abdominal sympathetic, in rabbits in which chronic lead-poisoning had been experimentally produced. He also found similar changes in a case of saturnism in man. The cœliac plexus was surrounded by, and infiltrated with, connective tissue, and the sympathetic cells were in part atrophied, but the fibres were little changed.

instance, diminished sensibility to touch in the back of one forearm and the front of the opposite leg, and, in another case, anæsthesia around the anus. Such symptoms are sometimes the result of neuritis. Muscular "stiffness" may be associated with the pain, and now and then there is painful cramp in the calves. When the pains have a well-marked neuralgic character in the arm or leg, they may follow the course of the nerves, and tenderness may accompany them. Epigastric pain has been already mentioned.

2. *Local muscular paralysis* with wasting is a very common consequence of lead-poisoning. It presents two forms. In one, the most frequent, loss of power precedes wasting, and the muscles present the "degenerative reaction," *i. e.* loss of faradic irritability (from acute degeneration of the nerve-fibres) and preservation of the voltaic irritability of the muscular tissue, just as in a traumatic lesion of a nerve. The common seat of this form is the extensor muscles of the wrist and fingers, and "wrist-drop" results. In the other form of saturnine atrophic paralysis the weakness and wasting come on simultaneously and proceed *pari passu;* faradic and voltaic irritability are both lessened in proportion to the wasting, just as in progressive muscular atrophy. This form may affect any part, but is most common in the small muscles of the hand. Between these two, intermediate forms are sometimes met with, in which a slight increase and qualitative change in voltaic irritability accompanies a moderate diminution in that to faradism.

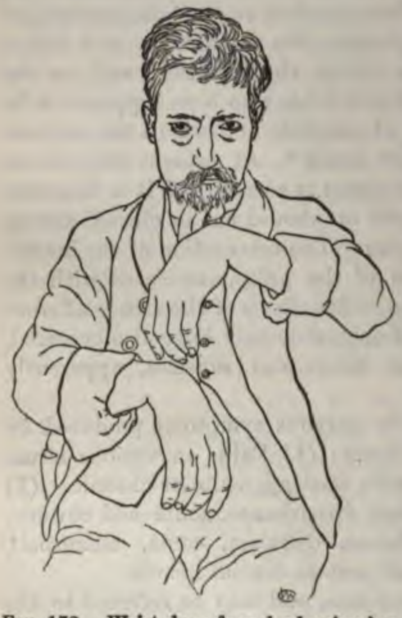

FIG. 172.—Wrist-drop from lead-poisoning. (From a photograph.)

In the acute form, not only is the wasting secondary to the weakness, but some muscles may be merely weak, and may recover without undergoing atrophy. In the muscles at the back of the forearm wasting is almost invariable. This extensor paralysis is usually bilateral, although one arm is often affected a little earlier than the other. The right arm is generally (but not always) the first to suffer, perhaps in consequence of the greater use of the right hand, since the left arm has been first affected in a left-handed man.[*] The interval between the affection of the two arms seldom exceeds two or three weeks; occasionally both are affected together. The paralysis reaches a considerable degree in a few days or a fortnight. The onset is more rapid in second than in first attacks, but it is never actually sudden. The first difficulty is in the extension of the fingers, often of the two middle fingers, sometimes of the first and second; but it soon extends to the others. The loss is that of extension at the metacarpo-phalangeal joints (by the long extensors); if the first phalanges are passively straightened, the distal phalanges can be extended without difficulty by the unaffected interossei and lumbricales. The thumb also suffers; extension of the phalanges (by the E. primi and E. secundi internodii pollicis) is lost, while its metacarpal bone can be still extended (or rather abducted) by the E. ossis metacarpi pollicis (Abductor pollicis of the Germans). The weakness of the extensors of the fingers is usually greatest towards the ulnar side; the first finger may be extended best, and the little finger least. Soon the extensors of the wrist become weak; sometimes the radial, sometimes the ulnar extensor suffers first, with a corresponding defect in the lateral movement of the hand, and deviation in the attempt to extend it. The hand ultimately "drops," and cannot be brought up to the level of the forearm. As long as the special extensors of the wrist retain power, this joint can still be extended when the fingers are flexed so as to close the fist, although the wrist cannot be extended when the fingers are also extended. The reason for this seems to be physiological. When in health the hand and fingers are both extended; the special extensors of the wrist act very little if at all; the movement at the wrist-joint is effected by the long extensor of the fingers. If, however, the fingers are flexed, the extension of the wrist is effected by the special extensors. These facts may readily be verified by placing a finger on the tendon of the E. carpi ulnaris or radialis during these movements. When the two muscles are paralysed the wrist cannot be extended, even with the fingers flexed.

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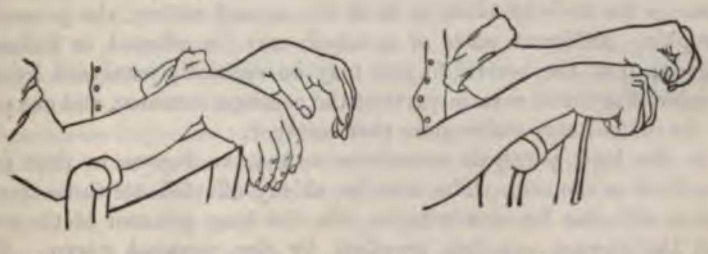
[*] Bernhardt, 'Deut. Arch. f. Kl.,' Band xxii, 363.

The flexors of the fingers are unaffected. Nevertheless their action is seriously impaired by the inability of the extensors of the wrist to

FIG. 173. FIG. 174.



FIGS. 173 AND 174.—Wrist-drop from lead-poisoning. Fig. 173 shows the maximum extension of the wrists and fingers; Fig. 174 the extension by the proper extensors of the wrist when the fingers are flexed.

co-operate with them, and to maintain the extension of the carpus necessary for forcible flexion. Hence flexion of the fingers flexes also the wrist, and the course of the tendons is thus so shortened that the maximum contraction of the flexor muscles exerts but little force. The repeated flexion of the carpus produces a slight displacement backwards of the carpal bones, and probably also a distension of the synovial sacs connected with them, so that a prominence forms over the carpus on the dorsum of the hand. It often alarms the patient, but is of no real consequence.*

In most cases of wrist-drop the paralysis is limited to the muscles mentioned—the common extensor of the fingers, the extensor indicis, the extensors of the phalanges of the thumb, and those of the wrist. All these muscles are supplied by the musculo-spiral nerve. But the supinator longus, also supplied by that nerve, almost always escapes, and so also, as a rule, does the extensor of the metacarpal bone of the thumb.

The affected muscles rapidly waste, and the back of the forearm becomes flattened, rendering the integrity of the supinator longus still more striking by contrast. The electrical reactions, as already stated, are those of nerve-degeneration (see vol. i, p. 54). The irritability to voltaism is for a time increased, and is commonly changed in quality. Such a qualitative change may even precede the onset of the paralysis (Erb). In an early case, in which one arm only was affected, I found in it the increased anodal contraction; while in the other, which was not paralysed, a distinct change was the occurrence of tetanic contraction with an unduly weak current. The excessive degree of voltaic irritability passes away in the course of a few months, but some irritability remains for a year or two, even when there is no return of power, and it can often be quickly increased by sedulous electrical treatment.

Although the atrophic palsy is usually limited to these muscles, it occasionally invades the upper arm. The deltoid suffers most frequently, and sometimes before the forearm muscles. Occasionally the biceps, deltoid, and brachialis anticus suffer, the palsy then assuming the "upper-arm" type. Very rarely the supinators suffer early. Occasionally the small muscles of the hand are involved, but it is more common for their affection to be of the second variety, the primarily atrophic. Different parts of a muscle may be affected in different degree; thus the several fingers may be variously paralysed by the greater affection of certain parts of the common extensor, and one part of the deltoid may suffer more than another.

In the legs, paralysis sometimes occurs, analogous to that just described in the arm. The muscles chiefly affected are those homologous with the forearm muscles, viz. the long extensor of the toes, and the peronei muscles, supplied by the peroneal nerve. The tibialis anticus, although supplied by the same nerve, usually escapes, like the supinator longus in the arm. The affected muscles present the degenerative reactions already described. Slight deformity of the foot, talipes equino-varus, may result.

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* This swelling is sometimes termed "Gubler's tumour."

Occasionally muscles in which there is no wasting may be weak, especially the muscles of the upper arm and thigh (including the ileo-psoas). In such muscles there is often (as we have seen) a slight indication of the qualitative change in voltaic irritability.

The second form, characterised by primary atrophy, occurs especially in the intrinsic muscles of the hand, but is sometimes extensive and irregular in its distribution, affecting many muscles in all four limbs. The wasting is slow, and accompanies, instead of succeeding, loss of power. Fibrillary twitchings are common, as in progressive muscular atrophy. The muscles still act to faradism, although the irritability is lowered in proportion to the wasting; the voltaic irritability is usually lowered in a similar manner, but it is sometimes a little greater than the faradic, and there is often a qualitative change. This form usually accompanies the degenerative extensor paralysis, but it may occur alone. It is much more obstinate than the degenerative form, and often persists long after the latter has recovered; it may, indeed, be permanent. For instance, a gentleman, poisoned by drinking-water, suffered complete wrist-drop on both sides, with wasting and loss of faradic irritability in the muscles, the voltaic irritability being preserved. There was transient weakness, without wasting, in the deltoids. The abductor indicis had slowly wasted with merely diminution of both faradic and voltaic irritability, and it remained in nearly the same state four years later, although the paralysis of the forearm muscles recovered in twelve months. An instance of extensive atrophic paralysis was afforded by a painter, aged thirty-two, with a well-marked line on the gums, and a history of three attacks of colic. First the right wrist "dropped," and then the whole arm became weak from the shoulder downwards. Four months later the left arm became paralysed in a similar manner, and soon afterwards both legs became weak. He came under treatment six months later; there was general wasting of the limbs, including in the arms the flexor muscles and supinator, the hand muscles being almost entirely gone. The muscles of the legs below the knees were much wasted. Everywhere faradic and voltaic irritability was lessened in proportion to the wasting, and even in the most wasted muscles, faradic irritability was not entirely extinct. The patient was treated with electricity, iodide, &c., for six months, but there was no discoverable improvement in the state of the limbs.

As the cases just mentioned show, the course of the primary atrophy is extremely chronic, and it has little tendency to recover. On the other hand, the degenerative paralysis, of which the wrist-drop is the type, usually recovers perfectly, although its duration is prolonged in proportion to the amount of wasting that has occurred. When this is great, the paralysis continues for six or eight, or even twelve months.

Voluntary power may return before faradic irritability. If the wasting is moderate, and faradic irritability is not entirely lost, recovery may occur in three or four months. Simple weakness without loss of faradic irritability usually passes away in the course of six or eight weeks. Wrist-drop readily recurs if the patient is again exposed to the influence of lead, and a very slight exposure is sufficient to bring it back. A painter had wrist-drop, and on his recovery became a college porter, spending his time in a small "porter's box." Some time afterwards a relapse occurred, and the only cause that could be ascertained was that the box had been repainted. Occasionally, a severe relapse is permanent.

There is usually little or no pain in the affected limbs, even at the time of the onset of the palsy. Rarely some sharp darting pains occur before the palsy develops, more often in the legs than in the arms. Sensation on the limbs is usually normal, but besides the limited impairment already mentioned, there is occasionally, with wrist-drop, a little defect of tactile sensibility in the area supplied by the radial nerve; in one case there were irregular areas of anæsthesia on the front and back of the hand and wrist, and on the tips of the fingers.*

Spinal symptoms have been observed in rare cases, especially general weakness of the legs, with a tendency to spasm, and ataxy with and without loss of the knee-jerk, similar to that which occurs in arsenical poisoning.†

Local Spasm.—Cramps in the legs are not uncommon in the early stage of the affection, but more definite local spasm is very rare. I have once known flexor spasm in the forearms to precede the onset of wrist-drop.‡ Another patient, with lead-poisoning and colic, but no paralysis, suffered from a brief but well-marked attack of tetany, for which no other cause than the lead-poisoning could be discovered; the paroxysms were confined to the arms, and recurred several times daily for about a week.

Tremor is less common in chronic poisoning by lead than in that by some other metals, but it is occasionally present in the hands in cases of long duration. It may be fine tremor like that of old age; occasionally it resembles closely paralysis agitans, more frequently it is increased by movement more than that of paralysis agitans, and is wider in range and more irregular in its distribution. I have known it to affect chiefly the flexors of the elbow and wrist and the supinator longus—the muscles which escape in paralysis. The lips and tongue may also be involved, and if there is no palsy the case then closely resembles one of mercurial poisoning.[*]

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* Oliver, 'Brit. Med. Journ.,' 1885.

† It has been suggested by Putnam ('Boston Med. and Surg. Journ.,' Aug., 1887) that slight weakness in the legs, with excessive knee-jerk, and also other symptoms of impaired nutrition of the nervous system, may be due to slight chronic lead-poisoning. He has found minute traces of lead in the urine of many patients thus affected. It is at present doubtful whether the detection of a trace of lead in the urine justifies us in ascribing any symptoms to lead-poisoning when there is no line on the gums. Of course, the condition of the gums must be taken into consideration in estimating the significance of the absence of a lead-line (see remarks in "Diagnosis").

‡ Similar transient spasm has been observed by Eisenlohr, 'Deut. Arch. kl. Med.,' xxxvi.

Local Saturnism.—Under this name, Manouvriez described[†] supposed local effects of lead on the parts to which it was applied. Such a local influence, except as regards the cutaneous nerves, is very difficult to understand, and the facts that have been supposed to demonstrate it have received very little confirmation. Manouvriez collected thirty cases in which local symptoms coincided with the local application of lead to the skin. In some there was a lead-line, in others there was not. In most cases there was loss of motor power; nine employed their feet to stamp on lead, and had weakness of the muscles of the legs. He ascribed the fact that wrist-drop occurs first in the right hand (or in the left in left-handed persons) to the chief use of the hand affected, and described two right-handed men, who manipulated lead with the left hand, in whom this hand suffered first. Among a large number of sufferers it would not be difficult to find some examples of such coincidence, even if there were no causal relation between the phenomena. Nevertheless some analogous facts have been also published by others. Monnereau[‡] observed in himself distinct loss of sensibility at the place where lead had been rubbed in. A smith, with colic and wrist-drop, who had worked with his left hand in lead, presented extensive anæsthesia in the left arm and none in the right. Again, a man, whose boots were strongly impregnated with white-lead, had anæsthesia of both feet, and weakness of some muscles of the legs. These facts suggest that lead may cause some local anæsthesia, but it is probable that the coincidence of muscular weakness has been accidental.[§]

Cerebral Disturbance; Encephalopathia saturnina (Tanquerel).—Symptoms of disturbance of the functions of the brain occasionally occur in severe lead-poisoning, and sometimes in cases of moderate severity. They vary much in character, and may be acute, subacute, or chronic. The disturbance is usually general, but in rare cases hemiplegic symptoms have been observed. The loss of motor power is slight, but that of sensation is considerable, and sometimes exists alone as partial or complete hemianæsthesia, involving general an special sensation. These symptoms are usually transient, and are apparently due to functional disturbance, such as occurs in hysteria. Such hemiplegia must not be confounded with that which results from cerebral hæmorrhage or softening, also common in the subjects of prolonged lead-poisoning. Transient hemianopia or amaurosis has also been met with.

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[*] Suckling, Tressillian, Stewart.

[†] 'Gaz. des Hôp.,' 1874, p. 290.

[‡] Monnereau, 'Thèse de Paris,' 1883; Capelle, ibid.

[§] Frémont ('La France méd.,' 1882, p. 892) has recorded a case of a n who one day worked with his left hand only in contact with lead, and did not h it for three hours. Next morning there was tingling in the hand and weak s of the extensor of the fingers. The possibility of a slight injury to the culo-spiral nerve renders this case of little significance.

Much more frequent is general cerebral disturbance, manifested by convulsions, delirium, and coma. Its onset is often acute, but may be preceded by certain slighter symptoms, giddiness, noises in the ears, tremor, restlessness, and insomnia. The convulsions may occur at the onset, or during the course of the delirium; they are epileptiform in character, consisting of tonic and clonic spasm. They are usually general, but occasionally one side is affected before, or more than, the other. Very rarely they may be hysteroid. The acute delirium often sets in suddenly, and is usually active, with considerable excitement, and sometimes attended with hallucinations of sight and hearing. When it is accompanied by muscular tremor, the condition may closely resemble delirium tremens. Somnolence and coma usually succeed delirium and convulsions. The coma is not often absolute; the patient can be roused for a few moments, but quickly relapses into unconsciousness. This acute cerebral disturbance is often accompanied by optic neuritis, and is frequently fatal, especially when there are repeated convulsions or deep coma. Slight fever (100°—101°) may attend these cerebral symptoms. Death may occur in the coma from respiratory failure, the heart continuing to act after the respiration has stopped. Occasionally such cerebral disturbance, especially convulsions, or coma-like unconsciousness, occurs as a transient symptom, and, like the amaurosis above mentioned, seems to be due to an effect of the poison on the brain much slighter than that which gives rise to the severe symptoms.

Chronic general cerebral disturbance also occurs. It may succeed the acute stage, or may commence gradually. I have seen several cases in which convulsions, similar, in character and course, to those of ordinary epilepsy, occurred in the subjects of lead-poisoning, without other symptoms, and continued for years after the toxic influence had ceased. Hysteria is often evoked by lead-poisoning in young girls, who are so often the subjects of its acute form.

More frequent, however, is mental disturbance, which may necessitate confinement. The most common form is melancholia with delusions, or mental derangement with hallucinations, somewhat resembling alcoholic insanity. Sometimes there are symptoms of mental failure and muscular weakness, resembling those of general paralysis of the insane, but coming on more rapidly. True general paralysis of the insane, with exalted delusions, typical course and post-mortem appearances, has occurred in the subjects of lead-poisoning, and may have been due to it (Monakow, Ullrich).

Laryngeal Palsy is a very rare effect of lead-poisoning. One or both vocal cords may be paralysed, and their muscles may be found pale and atrophied. The muscles affected have been the arytænoideus, all the intrinsic muscles of one side, and the posterior crico-arytænoids.*

Ocular symptoms are sometimes met with. The ocular muscles seldom suffer, but paralysis of the third nerve has been described by Buzzard. Inequality of the pupils has also been observed. Vision may be affected without any ophthalmoscopic change; there may be complete bilateral loss of sight, lasting many hours and resembling uræmic amaurosis, but occurring when there is no renal affection. When unilateral, or greater on one side, it may be associated with hemianæsthesia and affection of the other special senses on that side, and sometimes there is a concentric limitation of the field and loss of colour vision as in hysterical amblyopia. I have once met with transient green vision.

In cases of acute lead-poisoning, especially with cerebral symptoms, optic neuritis is frequent. The swelling of the papilla is considerable in degree, and accompanied by hæmorrhages ('Medical Ophthalmoscopy,' plate vii, fig. 6). In less severe cases I have occasionally seen slight chronic optic neuritis. The inflammation, both slight and severe, may pass off under treatment, without impairment of sight, but severe neuritis may cause "consecutive atrophy." Atrophy has also been described, without preceding neuritis, or with only transient congestion of the discs at the onset (Horner, Hutchinson). One eye may be affected before, and more than, the other. The ultimate condition is usually a greyish atrophy, often with white lines along the vessels. It may slowly progress to complete loss of sight.†

The several acute and chronic symptoms of lead-poisoning are often combined, and thus different cases vary much in character. Even in chronic cases there is often deviation from the common type. Colic, although the most frequent, is not constant; palsy or cerebral disturbance may come on without any preceding symptoms, especially in acute poisoning, and tremor or pains sometimes exist for a long time without paralysis. It is not practicable to draw any sharp line of demarcation between the symptoms of acute and chronic poisoning, or between the general classes into which we may roughly divide the symptoms.

PATHOLOGICAL ANATOMY.—Lead may be found in various organs by chemical analysis, but its presence only causes visible signs where it has been exposed to the influence of sulphur in a form that can enter into combination with the lead. The intestine may be dotted with minute black specks, as Fagge has pointed out. The chief lesions in lead-poisoning have been found in cases of local paralysis and wasting.

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* Morell Mackenzie; Seifert (three cases), 'Berl. kl. Wochensch.,' 1884. Horses working in lead mines are said to get laryngeal palsy.

† Retinitis, like that of kidney disease, but without albuminuria, has been described by Lehmann.

The wasted muscles are small and pale, sometimes even yellowish in tint, and are brittle. In cases of short duration, many of the muscular fibres are narrower than normal, but the striæ are preserved, although a tendency to longitudinal or transverse fissuring is sometimes seen. The nuclei of the sheath are increased in number. When the wasting has lasted for a considerable time, the fibres present still greater narrowing, a colloid or vitreous aspect, and there is often granular or fatty degeneration, although in some cases this is absent. The nuclei are still more increased in number, and there is also an increase of the connective tissue, in which fat may be found. The process of destruction goes on, until many of the sarcolemma sheaths become empty, and in the end, almost all trace of muscular tissue may disappear, connective tissue, nuclei, and masses of fat taking its place. The walls of the vessels often become thickened.

In the nerves, morbid changes are constantly found. They are always intense in the intra-muscular twigs, and in the branches from which these twigs proceed. They are usually considerable in the larger trunks, but commonly become slighter the further from the periphery the nerve is examined. Sometimes a few degenerated fibres can be traced throughout the nerves. The affection of the nerve seems to begin by a change in the medullary sheath, which becomes narrower, cloudy, and granular, the axis-cylinder being intact, and the outer sheath and nuclei presenting little alteration. With this, however, is combined a more considerable change, resembling the "Wallerian" degeneration that is secondary to a lesion of a nerve (see vol. i, p. 49). The myelin undergoes segmentation, the axis-cylinder also breaks up, the nuclei and protoplasm of the sheath become increased, and the products of this degeneration accumulate in masses of myelin and granule corpuscles. The slighter change (which Gombault,* who first described it, termed "periaxial neuritis") is sometimes segmental, affecting short tracts of the nerve that are separated by normal portions; or the upper part of the degenerated region may present only the change in the medullary sheath, while in the lower part there are the more extensive alterations. These changes are always most intense in the radial nerve and its branches; but it is only in extreme cases that all the fibres of the nerve are involved; generally, normal fibres are mingled with those that are degenerated, and become more numerous the higher up the nerve is examined. In a case in which the loss of power was limited to the parts of the extensor for the third and fourth fingers, Eichhorst found degeneration only in the corresponding branches of the radial nerve.† In some cases of long duration, appearances have been seen suggestive of a process of regeneration; these are very narrow but otherwise perfect nerve-fibres, or an axis-cylinder seems to end suddenly, and to be continued by two smaller ones (Westphal, Gombault).

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* 'Arch. de Phys.,' vol. v, p. 592.

† 'Virchow's Archiv,' 1890.

In most cases the degeneration ceases long before the anterior roots are reached, and these are normal. In a few cases, however, marked changes have been found in the anterior roots, similar to those in the peripheral nerves. Normal fibres were mingled with those that were affected. In one case of unilateral palsy, only the roots on the side corresponding to the paralysis were affected. The posterior roots have always been found unchanged.

In the spinal cord, in most cases, no morbid appearances have been found, even when wrist-drop has been of long duration and the neuritis well-marked. In cases with advanced kidney disease the walls of the vessel have been thickened, and sometimes there has been a general increase in the connective tissue throughout the cord. In a few cases, however, distinct alterations have been seen in the grey substance, especially in association with the slower form of muscular atrophy. In one case the consistence of the cervical enlargement was reduced. Atrophy of the ganglion-cells of the anterior cornua has been several times noted,* slight in some instances, in others considerable, especially in the inner anterior group of cells. This was especially conspicuous in a case described by Monakow in which there was slow wasting of the deltoid, thenar, and hypothenar muscles, in addition to the common wrist-drop. Spots of softening in the anterior cornua and intermediate grey matter were found in one case by Oeller, in addition to wasting of some of the nerve-cells.

The brain has presented no marked changes even in cases in which the cerebral symptoms have been considerable. Slight traces of inflammation have been found in the membranes in a few instances, and in the case in which there were the characteristic symptoms of general paralysis of the insane, there was evidence of considerable inflammation both of the pia mater and of the external surface of the dura mater. In the optic papilla the usual signs of inflammation have been found, and in Oeller's case there was also "hyaline degeneration" of the walls of the vessels, extending back into the trunk of the optic nerve. Degeneration of the larger arteries is common in cases of long duration, in which the kidneys are diseased, and resulting softening is often found.

Of the other organs, changes in the liver and kidneys are constant. In acute poisoning there is granular degeneration of the cells of each organ, and an accumulation of lymphoid cells between the rows of hepatic cells, and around the glomeruli of the kidney; increase of the interstitial tissues, and often contraction, are met with in older cases.

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* By Vulpian, Monckton, Zunker, Monakow, Carriew, Oeller, and others. Pigmentation of the cells has often been seen, but its significance is doubtful. I have seen it in one young subject.

spinal form. Rarely there seems to be a subacute process in the grey matter, extensive in distribution.*

The cerebral symptoms of lead-poisoning may be independent of any visible change. Although they have been ascribed to some vascular disturbance, it seems more probable that they are due to the direct action, on the nerve-elements, of the lead or of organic poisons which result from the imperfect action of the liver and kidneys. Headache, for instance, has been observed to coincide strictly with diminished elimination of urea (Oliver). At the same time, acute disturbance, although it begins in the nerve-elements, is apt to involve also the interstitial tissue, and even the vessels, and may assume the character of general inflammation in tissues disposed to this process. The optic neuritis may be such a direct effect of the poison, but it is possible that an irritative process descending from the brain aids in its production. The certain effect of lead on the peripheral nerves of the limbs makes the occurrence of this inflammation more intelligible, and also the paralysis of other cranial nerves occasionally met with.

The connection between lead-poisoning and gout was formerly thought to be simple. The poison lessens the elimination of uric acid, and the excess of uric acid in the system was regarded as the sole element in gout. But many facts (including the infrequency of gout among the subjects of lead-poisoning in the North, noted by Oliver) have led some authorities on the subject to regard the excess of uric acid as only one element in gout, and to consider that there must be other organic poisons in the blood in this disease, due to imperfect change in the substances that enter the blood, and should be excreted. An altered state of nutrition of the tissues may co-exist. This opinion is certainly supported by the wide diversity in the manifestations of gout, its remarkable effects as an inherited diathesis, and its distinct connection with several forms of rheumatism.

Diagnosis.—The recognition of lead-poisoning depends, first, on the character of the symptoms of nerve disturbance; secondly, on the existence of other indications of the presence of lead in the system; thirdly, on the discovery of the fact that lead is entering the system; fourthly, on the fact that lead is leaving the system by the urine.

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* Whether lead has also an action on the upper segment of the motor path (see vol. i, p. 176) so as to cause weakness without wasting and with an increase in myotatic irritability, we do not know. The facts collected by Putnam (p. 952, note) suggest that it may be so, but no distinct sclerosis of the pyramidal tracts has yet been observed, even in cases of long duration. It is, however, probable from analogy that the extremities of these fibres would be the first to suffer, and the fibres themselves might undergo as little change as do those of the upper parts of the peripheral nerves. It is evident that the excess of myotatic irritability can only be revealed where there is integrity of the lower segment. (Compare remarks on lateral sclerosis in "Progressive Muscular Atrophy," vol. i, p. 492.)

The diagnosis can only reach a high degree of probability when two or more of these indications are combined. It is especially important that too much weight should not be placed on the character of the symptoms alone; many errors in diagnosis are due to this.

When the symptoms are such as to suggest that lead may be their cause, the next step in diagnosis is to ascertain if there is any other evidence of the presence of this poison in the system. The variations in the lead-line have been already mentioned. If no line is conspicuous, and the gums are in general good, a search should be made for any place where the gum is not closely attached to the tooth, and the projections between the teeth must be carefully examined, since the line may be found only at these places. A fragment of distinct line is quite conclusive, and the absence of any line does not exclude lead-poisoning if the gums are everywhere in perfect contact with the teeth. On the other hand, if the gums are separated from the teeth in many places, and there is tartar on the teeth, the absence of a line, according to present facts, makes it in the highest degree improbable that the symptoms are due to lead. The only condition in which I have seen a line quite like that of lead, is silver-poisoning, but this is sufficiently distinguished by the tint of the skin.

The presence of a lead-line cannot be taken as absolute proof that the symptoms are due to lead. The line may last long after the poison has ceased to be active. Iodide of potassium removes lead from its organic combination in the tissues, but has no action on the deposit of sulphide of lead in the gums. Indeed, a solution of iodide of potassium is absolutely incapable of dissolving sulphide of lead, even if the two are boiled together. The lead-line does slowly disappear in the course of years, but I have known it to present little change in aspect, two years after the entrance of lead into the system had been stopped. Yet it has been observed to pass away in three months, and hence the precise state in which the pigment exists in the tissue of the gum probably varies. The persistence of the line is perhaps related to its previous duration.

Other effects of lead-poisoning, colic, gout, anæmia, seldom do more than corroborate the diagnosis; although, in the absence of a lead-line, owing to the intact state of the gums, frequent attacks of colic are of significance, and even the fact that gout has developed, without its usual causes, may be allowed some weight. In these cases, however, the diagnosis depends on the discovery of a source of lead-poisoning, or of the fact of the excretion of lead. If there is no obvious source of poisoning, the water and its receptacles should be examined. Most cases of lead-poisoning that are not due to occupation are due to the contamination of water, although the other occasional sources should always be borne in mind. The examination of the urine is often of much help of the investigator; it should be analysed after iodide of potassium has been taken for a week. It is [illegible] whether the presence of a very faint trace of lead is of significance. On the other hand, after iodide of potassium has been taken for several weeks, the absence of lead probably does not exclude lead-poisoning.*

It is on the above points that the differential diagnosis from maladies that resemble those produced by lead, chiefly turns. The wrist-drop has been confounded with paralysis of the musculo-spiral nerve, but this is always unilateral and usually comes on suddenly. In alcoholic neuritis the palsy may closely resemble that from lead, but the diagnosis is seldom difficult; the other signs of lead-poisoning are absent; pains are generally obtrusive; the legs suffer earlier, and there is almost always a clear history of alcoholic excess. Wrist-drop may be produced by arsenical poisoning, but in this also, the legs suffer before the arms, and sensory symptoms are more prominent. The greatest difficulty is presented by some cases of central disease. Progressive muscular atrophy (ultimately extensive) may commence by a subacute palsy of the extensors of the wrists exactly like that produced by lead, and atrophic palsy due to poliomyelitis may have the same distribution.† If the gums are perfect, an examination of the drinking-water or urine may be indispensable for the diagnosis. The cases of general muscular atrophy of saturnine origin scarcely ever present any difficulty, because they usually supervene on severe lead-poisoning that has caused characteristic symptoms.

Most errors in diagnosis occur in cases in which the nervous symptoms are anomalous and slight, and are not such as to suggest their cause. In the case of a lady with saturnine neuralgia, the cause of the symptoms might never have been discovered (for the perfect gums told no tale) had not her husband suffered from wrist-drop. A gentleman had some obscure cerebral symptoms with inequality of pupils; a fragment of lead-line opposite one tooth suggested lead-poisoning, and it was found that his water was stored in a leaden cistern. It is not possible to avoid error in such cases except by the habit of remembering lead as a possible cause in obscure cases. The severe cerebral disturbance is practically confined to workers in lead, and although the subjects have often not been long engaged in the occupation, the effect is usually due to a quantity so large as to cause other symptoms that cannot be mistaken.

Prognosis.—It may be said, generally, that the more acute the onset of paralysis, the better is the prospect of complete recovery. The acute cerebral symptoms are alone attended with immediate danger to life, and the prognosis is worse when there are convulsions, and especially when there is coma, than in simple delirium. Convulsions are of graver significance when they succeed delirium than when they occur first. Alcoholism increases much the gravity of the prognosis, and so do a considerable diminution in the amount of urine, marked, and especially acute, anæmia, and the evidence of organic changes in the kidneys. If death does not take place, the patient usually recovers completely. The prognosis in the chronic cerebral symptoms is unfavorable to recovery when these take the form of pronounced mental derangement or of epilepsy. Of the paralytic affections, those with the wasting and the degenerative reaction in the muscles, of which the wrist-drop is the type, almost always ultimately pass away, but their course is very slow, and the chief prognostic indications have been mentioned in the account of the course of the disease. In relapses, if these are due to a considerable dose of lead, the prognosis is less favorable, although two or three successive attacks may be recovered from. In all cases the ultimate prognosis must be influenced by the extent to which the patient can be withdrawn from the deleterious influence, but it must be remembered that, in chronic cases, the chief danger to life is often from the associated disease of the kidneys, with or without gout, and from arterial degeneration These may slowly increase, and even first manifest their presence, long after the lead-poisoning has ceased.

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* See note on next page. On the danger of mistaking bismuth for lead in the urine see Putnam, 'Bost. Med. and Surg. Journ.,' 1883, Oct. 14th, p. 315.

† As in the conclusive case mentioned on p. 896.

TREATMENT.—The first step is to arrest the entrance of lead into the system, and the second is to promote the elimination of the lead that has been already taken in. The chief agent for securing the latter is iodide of potassium, given in doses of three or five grains. Although iodide of lead is an insoluble salt, it has been abundantly demonstrated that the administration of iodide of potassium increases the excretion of lead by the urine, apparently in some complex combination. The amount of lead in the urine increases for a few weeks and then gradually subsides.* The quantity of lead excreted is much less than might be expected; this may be due in part to the fact that much passes away by the liver, but probably its chief cause is that the amount retained in the tissues, which the iodide liberates, is never actually great. When there are acute symptoms, and lead is circulating in the blood, iodide should not be given at once, since the toxic effects may be intensified by the sudden increase of the amount that is in the blood. Such a result has been noted by many observers. By saline aperients (especially the sulphates of soda and magnesia with some free sulphuric acid) the alimentary canal should be first cleared of any lead it may contain, or which may be excreted from its mucous membrane. If colic exists, it may be necessary to give a full dose of opium, to relax the spasm, before aperients can be effective. The bowels sometimes act spontaneously after a few hypodermic injections of morphia. Subcutaneous injections of atropine have been recommended for the same purpose. In two or three days, the administration of the iodide may be commenced. Sulphur baths have also been extensively employed to promote the elimination of lead, but their utility is doubtful. It seems that lead passes off by the skin only when the amount in the blood is very great, since, in chronic cases, even the copious diaphoresis produced by pilocarpine, does not eliminate the metal. If there is anæmia, iodide of iron may be advantageously combined with the iodide of potassium.

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* Thus, in one case in which no lead could be found in the urine before the treatment, five grains of iodide were given four times daily, and on the first day five milligrammes of lead were found in the urine, on the fourth 12, on the fourteenth 22, and then the quantity gradually lessened, and after two weeks more, only traces were found. (Swete, 'Brit. Med. Journ.,' 1882.)

For the local paralysis, strychnine has been given internally. Its mode of action suggests that it may do good, although its actual effect is difficult to appraise. There is no evidence that its local injection has special advantages. The most important agent in the treatment of the paralysis is electricity. The nerves have lost irritability, but the muscles still respond to the voltaic current, slowly interrupted, which certainly keeps up their irritability, and must therefore influence the nutrition of the muscles while the nerves are recovering. Thus the amount of muscular change is lessened, and the ultimate recovery is rendered more perfect. In a severe case, if the muscles are left alone for a few months, or are merely treated with faradism, there may be only the feeblest response to the voltaic current, but (as I have seen) this increases notably after two or three applications, and the paralysis, before stationary, may in a fortnight be distinctly less, and the improvement may steadily go on to complete recovery. In such a case the value of electricity scarcely admits of question. Only the voltaic current, to which the muscles respond, is of service. Faradism should not be used unless it excites contraction, as, for instance, in the primary atrophic form. But in this it is difficult to get evidence of the value of the application. Massage of the limbs is also of service. Cerebral symptoms, especially convulsions, are often associated with increased tension of the pulse, and it is said that nitrite of amyl has a marked effect upon them (Oliver). Nitro-glycerine, by the mouth, would probably be useful in such cases, and might be combined (in alcoholic solution) with the acidulated saline aperient.

ARSENICAL POISONING.

Arsenical poisoning differs from lead-poisoning in being seldom the
Disturbance of the nervous system has generally
se, taken by accident or design, or
ts containing arsenic;

few days to a few weeks; among recorded cases are intervals of six days, two, three, and four weeks.[*]

The paralysis in the legs and arms has the same distribution as that commonly produced by lead-poisoning, but sensory symptoms are much more pronounced. Atrophy of the muscles occurs very rapidly. The electrical irritability presents the same degenerative changes as in other toxic palsies, but in very severe cases the voltaic irritability may quickly fall below the normal. The knee-jerk is often lost. Sometimes other muscles are involved, those of the upper arm and thigh may lose power, but not to the same degree as those of the extremities. Very rarely the trunk muscles have also suffered. Paralysis of accommodation has been observed.[†] Muscular tremor has been an occasional symptom, and acute palsy has been preceded by cramp-like contraction in the limbs.

Arsenical ataxy, although less frequent than the muscular paralysis, is not rare. It is usually met with in the legs, but has been observed also in the arms. It resembles closely the inco-ordination of tabes, but develops in a much more acute manner, and is accompanied by considerable muscular weakness, and sometimes by the characteristic atrophic palsy in the muscles in front of the lower leg. It has, however, been known to come on (or become evident) as the palsy was passing away. Severe pains attend the onset, and there are also subjective sensations, formication, tingling, &c. The sensibility of the muscles is lessened or lost, and so, as a rule, is the knee-jerk; sometimes it is preserved, and in the case of poisoning from muslin it was excessive, in spite of atrophy below the knee. The loss may last longer than the other symptoms, and has been known to continue for a year.

The other sensory symptoms are like those met with in alcoholic neuritis. They occur first in the legs and then in the arms. Various subjective sensations may be experienced at the onset. Pains are constant, in chronic cases they are at first slight, but in all they become, sooner or later, sharp, darting or burning in character, sometimes momentary. They are felt in various parts of the extremities, and often take the course of the nerves, in which there may be marked tenderness. At first there is hyperæsthesia of the skin, subsequently, in many cases, loss of sensation to touch or to all forms of sensation. Painful impulses, when not lost, may be delayed. Hyperalgesia may coexist with loss of tactile sensibility—the "anæsthesia dolorosa." The loss usually begins in the foot, and may first involve the sole, spreading gradually over the foot and lower part of the leg, above the ankle. In the hand similar symptoms occur later and in slighter form. The tactile loss may first occur on the fingers and palm, and be greater in one nerve area than in another. When sensation is impaired on the sole the plantar reflex is lost. The knee-jerk may persist, but is often lessened or lost, always when there is any atrophy of the extensors of the knee.

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[*] See Dana, 'Brain,' vol. ix, [illegible] 1880; Gerhardt, 'Virchow's Jahresb.,' 1882; S [illegible]

[†] Marik, 'Virchow's Jahr [illegible]

Cutaneous eruptions are sometimes observed, and seem to be the direct effect of the poison on the nutrition of the skin, often doubtless produced through the nerves, but they may be disconnected with the other neuritic symptoms. The hair may fall off or become grey, the nails may present changes and even separate, bullous and erythematous eruptions may occur, and herpes zoster is especially common, generally on the chest. It often follows the medicinal use of arsenic, when there are no toxic symptoms, as Hutchinson first pointed out. Brownish pigmentation of the skin is also both common and characteristic. In severe poisoning it occurs in roundish spots, which sometimes first lead to a suspicion of the cause of other symptoms. The prolonged medicinal use of arsenic usually causes a finely punctate pigmentation. Optic neuritis has been once observed (Dana). The pulse is often frequent, and sometimes the temperature is raised.

Pathology.—The dependence of the symptoms on changes in the nerves, suggested by Jäschke in 1882, was rendered practically certain by the discovery of the cause of the similar symptoms that are produced by alcoholism, and has since been proved. Characteristic degeneration was found by Erlicki in the radial and peroneal nerves. The cells of the anterior cornua in the cervical and lumbar regions of the spinal cord also presented slight changes. The chief effect of arsenic is doubtless always to cause a degenerative parenchymatous peripheral neuritis. In some cases the chief action is on the motor nerves, giving rise to the atrophic palsy, in others on the sensory nerves of the skin, producing the subjective sensations and sensory impairment, or of the muscles and joints, causing the ataxy. Paralytic symptoms, or sensory and ataxic symptoms, may predominate in different cases, but are not met with exclusively in any, and in severe cases both classes are met with in considerable degree.

Diagnosis.—The distinction from lead-poisoning rests on the common onset after acute symptoms, on the more pronounced disturbance of sensibility, the early affection of the muscles of the lower leg, and on the absence of other indications of the effect of lead on the system. In chronic cases the discovery of a source from which arsenic may have entered the system is, as a rule, possible, and its presence may be confirmed by an examination of the urine. The absence of a history of intemperance is the most important general distinction from alcoholic palsy, which that produced by arsenic closely resembles; but the most marked peculiarities are the deferred onset after acute poisoning, the manner in which ataxy may follow paralysis, and the long duration of the effects of a single ingestion of the poison. In chronic poisoning the early pains are sometimes ascribed to g rheumatism; in all cases in which there are persistent pa limbs, a careful search should be made for tenderness of the nerve-trunks, which points to the probability of toxic irritation.

The course of arsenical palsy is similar to that which is due to lead, and the treatment necessary is essentially the same. Iodide of potassium appears to have a similar influence in removing the arsenic from the system.[*]

SILVER-POISONING; ARGYRIA.

Chronic silver-poisoning is due almost exclusively to the medicinal use of the metal. It is less frequent now than when a long course of silver was the fashionable treatment for epilepsy and spinal-cord disease. Most cases are due to internal administration, but argyria has been known to result from applications to the palate or tongue, continued during many years. Working in silver involves no liability. Of the cases that have come under my own notice, two were epileptics (in whom the fits continued in spite of the influence of the silver), a third had been treated with silver for a syphilitic gumma pressing on the spinal cord, and a fourth had taken a salt of silver in a "dinner pill." The discoloration of the skin, which is the characteristic of 'argyria,' depends on a deposit of silver beneath the Malpighian layer,[†] and commences when about an ounce of a salt of silver has been taken, during any period of time. It is accompanied by a black line on the gums close to the tooth, closely resembling the lead-line. This appears before the darkening of the skin, and is therefore important as a warning.[‡] Among other internal organs,[§] silver is deposited[||] in the kidneys—in the Malpighian bodies, and around the tubules; albuminuria has been observed both in animals and in man. In these two points the effect of silver resembles that of lead. In animals, moreover, paralysis is produced. I have been unable to find any recorded instance of paralysis in man, but one remarkable case has come under my own observation, which shows that the effect of silver may be almost the same as that of lead. The case was that of a gentleman, aged forty-four, who had been ordered, twelve years before, a "dinner pill," without any caution or other direction than that one pill was to be taken three times a day. He did not follow the directions literally, but took sometimes one, sometimes three, a day, for a few days at a time. He estimated that he had taken about six pills a month during the twelve years. After he had taken the pills for about eleven years, a dusky tint of the face attracted notice, but its nature was not suspected until the tint had become well marked, when it was found that the pills contained silver (the exact form could not be ascertained). Two months before I saw him, the extensors of the right hand became weak; shortly afterwards, those of the left. When he came under observation the tint of the skin was very characteristic; his gums presented a well-marked black line, which resembled that of lead in being present where there was tartar, and absent where the gum was in perfect apposition to the teeth. In both arms there was paralysis of the long extensor of the fingers and of the extensors of the phalanges of the thumb, not of the extensor of the metacarpal bone of the thumb. On the right side there was also paralysis of the radial extensors of the wrist. The affected muscles were wasted, and their irritability to faradism was lost; that to voltaism was preserved and increased. There was no affection of the legs. The urine contained some albumen and casts, and the patient had had several attacks of gout. During three months in which he remained under treatment (electricity, iodide of potassium, &c.) there was slight improvement in the arms, but no considerable change. He soon afterwards showed symptoms of cancer of the liver, of which he subsequently died, but no post-mortem examination was obtained.

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[*] A full bibliography is given by Dana, loc. cit.

[†] See Neumann, 'Wien. med. Zeitung.,' 1878, No. 10.

[‡] Respecting its constancy, see Chaillon, 'Thèse de Paris,' 1878.

[§] The distribution and precise seat in the internal organs were carefully investigated in one case by Weichselbaum, 'Allg. Wien. med. Zeit.,' 1878.

sid as an organic compound (Krysinski, 'Gaz. lekarska,' 1886, and 'ib., i, 372).

MERCURIAL POISONING: HYDRARGYRIA.

Chronic poisoning by mercury causes symptoms of which some are due to an action of the poison on the nervous system. These symptoms rarely follow the medicinal use of mercury, but occur chiefly in those who work in the metal. Mercury will volatilise at all ordinary temperatures, and it is probable that it enters the system mainly through the lungs, but it may sometimes pass in through the skin when certain forms of mercury are much handled, and want of cleanliness may increase the amount of mercury that gets into the system by causing contamination of the food. Sufferers are met with especially among those who work in quicksilver mines, at silvering mirrors, and in making barometers and thermometers, and also occasionally among bronzers, furriers, and hat-makers, mercurial salts being used for preparing the skins. The weakly suffer more readily than the strong, and the individual differences in susceptibility are very great. The symptoms occasionally develop after the exposure to the cause has ceased.

The nervous symptoms may exist alone, but sometimes they are preceded by others outside the nervous system, which resemble those of acute mercurial poisoning. These are stomatitis and ulceration of the mucous membrane of the mouth, with extreme fœtor, necrosis of bone, gastric and intestinal catarrh. The remarkable removal of lime from the bones and its deposit in the kidneys, discovered as a consequence of acute mercurial poisoning by Prévost and Fruchtiger, does not seem to attend the chronic form, in which the kidneys do not suffer.

A peculiar tremor, known among the workmen as "the trembles," and medically as "mercurial tremor," is the most common and characteristic symptom. It is at first occasional, occurring only when the patient is excited, and is always increased by emotion. It usually begins in the face and tongue, then invades the arms, and afterwards the legs. At first the tremor occurs only on movement, but ultimately it may become constant. In the former case, the condition of the patient resembles that of one suffering from disseminated sclerosis, except that the tremor is less wide and less irregular than in characteristic cases of the latter disease. When constant, the tremor resembles that of paralysis agitans. During sleep, the tremor usually ceases, but in extreme cases may only lessen. It interferes much with articulation, rendering the speech stammering and hesitating. When considerable it may render the movement of the arms so unsteady that the patient cannot feed himself, and his gait becomes affected. At first the limbs are strong, but after a time muscular power is impaired, sometimes more in one limb than in another, but it seldom progresses to complete loss. Rarely there is wide-spread weakness without tremor.* Reflex action and power over the sphincters are always unimpaired, and the electric irritability of the muscles is normal throughout.

Psychical symptoms are also common, and may precede the tremor. They have been very carefully studied by Kussmaul. Irritability, and a difficulty in giving attention to a subject, are often the first symptoms, and may be accompanied by considerable mental distress and sleeplessness. Hallucinations sometimes occur, and there may even be outbreaks of maniacal excitement, but the insanity rarely corresponds to any distinct variety. This condition has been termed "mercurial erythism." It is sometimes accompanied by headache and palpitation of the heart.

Sensory symptoms are present in many cases; pains, especially in the region of the fifth nerve and in the joints; formication in the limbs, and sometimes loss of sensibility to pain, with paroxysms of distressing sensations in the thorax, resembling those of asthma. The sensory disturbance always augments the mental instability. In rare cases more grave cerebral symptoms occur—considerable hemiplegic weakness, aphasia, and deafness.

Although the tremor may have been extreme, no alterations have been discovered in either brain or cord, even with the aid of the microscope. The character of the symptoms makes it probable that they are due to disturbance of the brain rather than of the spinal cord. In animals mercurial poisoning is said to cause degeneration of the medullary sheaths of the muscle-nerves, but not of their axis-cylinders.†

The diagnosis is rarely difficult. From paralysis agitans the tremor is distinguished by its immediate increase on movement, and by the absence of the rigidity of the limbs and fixation of the features. In disseminated sclerosis, there is never the peculiar tremulous stammering that is met with in mercurial poisoning, and the movements are wider in range and wilder in their irregularity. The symptoms often resemble those of general paralysis of the insane more than those of any other disease, but such preponderant tremor is rarely met with in general paralysis, and the inequality of pupil, optimism, and indications of spinal degeneration, present in the latter, are absent in mercurial poisoning. The presence of stomatitis should always direct attention to the probable nature of the disease, and when this is once suspected, the patient's occupation is, as a rule, decisive. The greatest diagnostic difficulty is presented by some cases of lead-poisoning in which tremor is the chief symptom. These are distinguished by the presence of the lead-line in the gums, and if there is a suspicion that the patient is suffering from both poisons, the question can readily be set at rest by an investigation into the materials with which he works. If these are free from mercury, the symptoms are certainly due to lead only.

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* Letulle, 'Arch. de Phys.,' 1889.

† Ibid.

The prognosis is in most cases favorable as to the ultimate issue, but all cases are prolonged, and in some the tremor never passes away. For recovery, it is essential for the patient to cease to work in mercury. No precautions avail to prevent sufficient absorption to maintain symptoms that have once developed. Nutritious food, fresh air, and iron are important aids to recovery. To promote the elimination of the mercury, iodide of potassium may be given, but in small doses —two or three grains very gradually increased. There seems no doubt that it does facilitate elimination, and the sudden administration of large doses has been followed by a considerable increase in the symptoms; apparently mercury released from the tissues passes into the blood and to the nervous system. Neither sweating nor sulphur baths seem to cause an elimination from the skin, although it is said that the latter have been found in some way to increase the amount excreted by the kidneys. The stomatitis is relieved best by chlorate of potash.

ALCOHOLISM.

The nervous system is especially sensitive to the influence of alcohol, and suffers in many cases, more than other tissues, from the effects of habitual alcoholic excess. The acute poisoning by alcohol to which, *par excellence*, the term "intoxication" is applied, is chiefly of physiological and toxicological interest. Only the morbid states that are distinct from simple drunkenness come into the province of medicine. These are exceedingly numerous; many of the organic and functional diseases already described may be produced, or predisposed to, by intemperance; some other derangements are due chiefly to this cause, and are seldom or never produced by any other. To these the term "alcoholism" is generally applied. These disorders and symptoms may be acute, subacute, or chronic. The acute disturbance generally takes the form of an attack of delirium, called, from the tremor that accompanies it, "delirium tremens."* The chronic derangements are exceedingly varied.

All forms of alcoholism have certain common causes. The tendency to drink is often inherited; less commonly it is related to a family disposition to suffer from definite diseases of the nervous system, especially from insanity.† It is probably facilitated by peculiarities of nervous constitution, in consequence of which the sensations of intoxication are more pleasurable in some persons than in others. Parental intemperance, moreover, often involves facilities for habitual excess. Occupation is notoriously influential, partly on account of the facilities for drinking afforded to those who are engaged in the distribution or sale of alcoholic liquors, partly on account of the social temptations to drink involved in other callings. The temporary relief to physical and mental depression afforded by alcohol is another potent cause of intemperance.

ACUTE ALCOHOLISM, ALCOHOLIC DELIRIUM, DELIRIUM TREMENS.

Alcoholic delirium occurs under two forms: as a primary affection, and in association with some local inflammation or injury. The latter may be termed "associated delirium;" it has a double causation, the influence of alcohol and the influence of a local disease. The latter may be sufficient by itself to cause some delirium, although far less in degree, and without the peculiar characteristics of the alcoholic form; or it may be wholly insufficient to derange the mind. The simple primary delirium varies much in the intensity of its symptoms; it has been divided into two classes according to the presence or absence of considerable fever, but the difference depends only on the degree of severity, and there is no real justification for the division.

CAUSES.—Delirium tremens is almost confined to drunkards, in the popular sense of the word. In this respect it differs from the "associated delirium," which is not uncommon among those who have habitually taken an excessive quantity of alcohol to keep up their energy against the depressing influence of debility or the prostration of some chronic disease. Thus a man who had never been known to be intoxicated, and was believed to be temperate, died with symptoms of acute delirium tremens; but it was found that his lungs were extensively diseased, and that he had been keeping up his working power by large doses of alcohol during the acute development of the pulmonary disease.

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* By Thomas Sutton ('On Delirium Tr

† Of 600 drunkards admitted int
intemperance could be traced in
in 168. Insanity could be t
established before twenty-f

Delirium tremens is far more common in men than in women, the proportion being about six to one.* There are no facts to show whether there is any sexual tendency to the disease, such as certainly exists in the case of another effect of alcohol, multiple neuritis, to which women possess a greater liability. As far as the figures given below suggest any conclusion, it is that drinkers in the two sexes are equally prone to suffer from delirium tremens. The disease is most frequent in the middle period of life, thirty to forty years. In both sexes the maximum mortality is at the same age, a little later than the maximum frequency, because second and third attacks are the most fatal; one third of the total number of deaths occur between thirty-five and forty-five years of age, and about four fifths during the twenty years—thirty to fifty. It rapidly lessens in later life, no doubt because the worst drinkers acquire the habit early, and die off from some effect of intemperance, but cases of delirium tremens occur even up to advanced senility. Moreover, females bear a proportion to males that steadily increases as life goes on, probably because drinking habits are acquired relatively later, under the influence of individual causes rather than as an effect of occupation or of social life.† Under twenty-five, the affection is chiefly met with as a complication—especially of pneumonia.‡ It is probable that an hereditary tendency to neuroses facilitates the occurrence of this disease, especially a tendency to intemperance,§ and insane heredity unquestionably modifies its course. The disease is less frequent in beer-drinkers than in those who drink wine, and most frequent in spirit-drinkers, but it is probable that the amount of alcohol taken has much greater influence than its form.

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* During the twenty-five years ending 1872, there died from this cause in England and Wales 10,448 males and 1398 females. It is probable that this disease is one on which the Registrar-General's returns are fairly accurate, since this cause of death is unlikely to be given unless there is a history of intemperance. The figures give the proportion of deaths as 7½ males to 1 female. But the disease is not only more common in males, it is also more often severe, and therefore it is more often fatal. Hence the disproportion between the sexes is doubtless rather less in cases than in deaths. Of 54 consecutive cases in University College Hospital, 8 were in females, giving the proportion of 6 to 1, which is probably very near the truth.

† The proportion of deaths of males to each female is about 12½ in the ten years—fifteen to twenty-five, and then steadily falls, the proportion in each successive later decade being 10—1, 7¾—1, 7½—1, 6—1, 4½—1, and only 3—1 in those over seventy-five.

‡ A few cases have been recorded of acute alcoholism in childhood. Thus a boy of five, to whom spirits, and also wine, had been given daily, had a brief but typical attack of delirium tremens after a fracture of the thigh (Cohn, 'Berlin. kl. Wochenschr.,' 1888).

§ In Germany neuropathic heredity was traced by Krukenberg in half the patients (265), and in one fifth the father was intemperate ('Zeitsch. f. kl. Med.,' xix).

In most cases the malady follows a severe "bout" of drinking, often without any interval. Occasionally there has been a cessation of drinking for a few days before the onset of the delirium. These cases have led to the idea that the discontinuance of the stimulant has been the cause of the attack; but it is more probable, as Anstie and others have suggested, that a sudden distaste for liquor was really the first effect of the commencing disease. The onset occasionally follows some slight injury or trifling illness, which apparently disturbs the nervous system, and serves to excite the disturbance that is ready to break out. Such cases present a gradation to the "associated delirium tremens," in which there is some real cause of general indisposition, a severe accident or a local inflammation, especially pneumonia. The pyrexia resulting might or might not be attended with delirium in the healthy; in the alcoholic, the delirium is severe, and presents the same characteristics as in the pure form of delirium tremens.

SYMPTOMS.—The symptoms of delirium tremens usually develop gradually but rapidly, attaining considerable intensity in the course of two or three days. Disturbed sleep, loss of appetite, and restless irritability are commonly the first indications of the impending disorder. Distressing or horrible dreams of peculiar vividness attend sleep, and the patient cannot shake off their effect when he wakes. He is depressed, uneasy, restless during the day, often annoyed with floating specks, or flashes of light before his eyes, and the next night brings the same distressing dreams. As soon as he closes his eyes, unpleasant scenes present themselves before him, and he tosses about all night, half awake, or perhaps is unable to obtain any real sleep. Next day, from time to time, more definite hallucinations of sight or hearing occur; the patient can shake them off, and knows that they are unreal, but they return again and again. When night comes, they take possession of him, prevent all sleep, and the following morning they are no longer to be shaken off. This insomnia, first partial and then absolute, with the distressing hallucinations which disturb the imperfect sleep that is obtained, characterises the early period of the disease. The transition to more considerable disturbance is usually attended with unnatural loquacity, and by indications of suspicion and of uneasiness. The period of definite delirium begins with the persistence of the hallucinations during the day. They are chiefly visual, sometimes auditory, and occasionally related to some cutaneous sensations; they are almost always unpleasant. At first the objects that the patient actually sees are transformed into other things than they really are, often into living creatures in active motion; the pattern of the wall-paper becomes beetles, spiders, snakes; and then these appear spontaneously, and are crawling about the bedclothes; the sufferer tries to brush them away or to escape from them. At first he can be recalled from his aberration for a few moments, and will answer questions correctly, but soon relapses into the delirium. Delusions of wider extent grow out of the hallucinations, or are excited by some bodily discomfort. Faces appear before him, grinning at him, or he fancies that persons are standing by his bedside, upbraiding or abusing him. A characteristic of the delirium is the versatility of the false ideas; they change continually. Gradually he ceases to be able to shake them off, even for a moment, and no longer recognises his friends or doctor. The attendants are thought to be the subjects of his delusions, and he may strike at them under the impression that they are attempting to injure him. Often the modified incidents of his daily life seem to occur before his eyes. He usually talks incessantly, but may wander incoherently from one subject to another in the course of the same sentence. Auditory hallucinations may occur early or may be grafted on those of vision, and the patient thinks he hears reproaches or insults from the fancied bystanders. Less commonly an unpleasant smell or a disagreeable taste annoys him, or he fancies that insects are crawling on his skin. The delusions often excite intense emotions of horror or dread.

The characteristic tremor is usually present from the first. It occurs only on movement, and is irregular, but considerable in range; it is most conspicuous in the arms, the face, and the tongue, but is to be seen also in the legs when these are put into voluntary movement, especially if the patient attempts to stand. It is the more conspicuous, because the patient is usually in constant movement, picking at the bedclothes, searching for imaginary objects, attempting to get out of bed. This extreme restlessness is seldom absent. Often, in addition to the tremor on movement, there are spontaneous, slight, partial, muscular twitchings, and in severe cases these may amount to considerable shock-like contractions, and may occur in the muscles of the trunk as well as in the limbs.

The countenance of the patient is often flushed, and the conjunctiva injected; but occasionally the face is pale, and sometimes initial flushing is replaced by pallor as the stage of depression comes on. The pulse is frequent and soft; the frequency is proportioned to the severity of the attack and to its duration. At first the pulse is full and large, but as the disease goes on, it usually becomes smaller as it gets more frequent. In severe attacks the frequency may be 140 or 150, sometimes even more, and it is then always feeble. The patient usually perspires freely. The temperature is raised in all except the slightest cases. When the attack is of moderate severity, the elevation is slight, and does not exceed two or three degrees, but in severe attacks it may reach 103°, 104°, or even 105°. Occasionally, hyperpyrexia is attained, 108° or 109°, a symptom of fatal significance. The tongue is thickly coated, and there is usually complete anorexia; the patient may or may not be thirsty; thirst is often a consequence of the copious perspiration. The urine is scanty if there is much sweating, and is usually deep coloured, of a high specific gravity, and very often contains a small quantity of albumen apart from organic kidney disease. There is occasionally slight blurring of the edges of the optic disc, with, according to Uhthoff, some opacity of the retina;* but, as a rule, the fundus is normal. Sometimes acuity of vision is lessened, or the fields, for a time, are restricted.

This state usually continues for two, three, or four days without interruption. The patient gets no sleep, or only dozes for an hour or so at a time, and wakes up unrefreshed. The pulse becomes softer and more frequent, and the muscular strength becomes lessened by the constant exertion. In favorable cases the patient at last falls into a sound sleep, which continues for eight, ten, or twelve hours, and he wakes up free from all mental disturbance, or with only a trifling amount of delirium, which another sleep entirely removes. The tremor lessens, but often continues in slight degree, sometimes for some days or weeks. The pulse falls in frequency and improves in strength, after the cessation of the delirium, and, in the course of a few days, the slight tremor and muscular weakness are the only relics of the attack through which the patient has passed. In other cases, however, sleep, when it does come, is briefer, and is not followed by the same signs of improvement; the delirium continues, but is less active than before, and the signs of general prostration are greater. In such cases there is considerable danger to life. A second or third period of sleep may bring improvement, and slow convalescence may ensue at the end of six or seven days, but in other cases the prostration increases, the pulse becomes still feebler and more frequent, and a comatose state comes on, in which the patient dies. This unfavorable condition is sometimes reached without any sleep having occurred, in spite, it may be, of strong narcotics. Convulsions or hyperpyrexia may attend the increased prostration of the later stage, and are generally a fatal omen. Convulsions may also occur at the onset, and are not then of much significance; at the later period of an attack, by the disturbance of the nervous system which they reveal, as well as by their effect on the patient, they add greatly to his peril. They resemble the attacks of ordinary epilepsy.

Complications.—The most important of these are other diseases due to alcohol, or local acute inflammations, especially pneumonia. Cirrhosis and fatty degeneration of the liver, and disease of the kidney, are especially serious complications. Uræmic coma occasionally comes on in the course of delirium tremens. Pneumonia is a very common accompaniment, and its occurrence is facilitated by alcoholism; it may lead the way, and the nervous disorder may be secondary in time, although ultimately dominating the symptoms, or the lung affection may be a subordinate complication of the disease, developing intemperance. Signs of actual acute meningitis are rare, but have been occasionally found in both the pia arachnoid and the dura mater. Congestion of the bases of the lungs is common, and sometimes there are signs of congestion in the kidneys, but the alterations met with outside the nervous system are chiefly the chronic changes that result from intemperance.

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* Uhthoff, quoted by Moeli, 'Charité Annalen,' ix, p. 524.

Alcohol may be found by chemical analysis in various organs of the body up to the fifth day after its ingestion; by the seventh day it has disappeared. But the presence of alcohol may be demonstrated, in like manner, in persons who have been drinking, but have not suffered from delirium tremens. We cannot, therefore, regard it as affording a full explanation of the disease. This evidently depends on an acute disturbance of the nerve-centres, probably involving all parts of them, but especially the cerebral cortex,—a disturbance of function produced by the toxic agent, running a definite course, with a tendency to subside, provided the continued action of its cause is prevented.

Diagnosis.—The disease is easily recognised. The preponderance of visual hallucinations, and the associated tremor, distinguish it from acute mania on the one hand and from symptomatic delirium on the other, while the absence of headache and of paralysis is a distinction from ordinary acute meningitis. Cases of general paralysis of the insane, in which muscular tremor is conspicuous, have been mistaken for delirium tremens; but if there is much mental alteration, its character is altogether different in the two diseases; the exaggerated delusions of general paralysis, and the mental complacency that commonly attends them, are very different from the anxiety and horror of the alcoholic. When depression occurs in general paralysis, the mental state has generally developed gradually, in a manner very unlike the acute onset of delirium tremens.

Prognosis.—The prognosis is good in first attacks, free from complications, and in those whose tissues are not seriously damaged. The pulse and temperature afford the most important indications. The danger is great in proportion to the frequency, softness, and small size of the pulse, and, as Anstie has shown, the sphygmographic tracing presents very clearly the serious characters, although the instrument is not actually needed to detect them. As a rule, the occurrence of sound sleep for six or eight hours greatly improves the prognosis, but if an exceedingly unfavorable pulse presents no improvement after sleep, the danger is great, and the patient is very likely to die in a few hours. A temperature above 103° is always of serious import, and the danger is greater in proportion to the degree of pyrexia; a rise to 105° and over is seldom survived. Moreover, a sudden considerable rise in the later stages of the disease, is also of very grave significance, even although the actual height attained is not extreme. Advanced years add greatly to the gravity of delirium tremens, especially when there is considerable pyrexia; in a man of sixty, a rise to 103° is as serious as would be a temperature of 105° in a young person. Any complication also lessens the chance of recovery, the most serious being pneumonia and kidney disease. If the pneumonia is double, death is practically certain. The prognosis is also grave when delirium tremens is associated with traumatic lesions, both because the tendency to depression and collapse is increased by the shock of the original injury or operation, and also because wounds run a very unfavorable course in this condition.

TREATMENT.—The mental state of the sufferer from delirium tremens necessitates very careful management. It is essential that all undue excitement should be avoided, and only the necessary attendants should be in the room. It is well to have the light dim, unless this distinctly increases the patient's suspicion and uneasiness. When there is much disposition to act according to the false ideas, male attendants are necessary, and physical restraint is often indispensable, lest the patient injure himself or others. He may even attempt suicide by jumping from a window or swallowing some accessible poison, sometimes deliberately, sometimes under the influence of a false idea. Hence windows should be securely fastened, and no lotions or poisonous liquid should be within the patient's reach. In many cases the patient can be kept in bed by judicious management and persevering persuasion on the part of the attendants. In other cases, however, force is needed. The mode in which it should be applied is a matter on which authorities differ. If there is great violence, continued exertion on the part of the attendant is necessary to keep the patient in bed; two attendants are needed to cope with a strong delirious man, and the constant struggles are exhausting to the patient. The "strait waistcoat" offers, in such cases, an effectual mode of restraint; the interference with the movement of the chest produced by it has been urged as an objection to its use, but this drawback has certainly been exaggerated. Leather gauntlets on the wrists and ankles may be secured to the sides and foot of the bed, and restrain the patient effectually without any interference with respiration. Bandages are sometimes employed for this purpose, but may cut into the skin if the struggles are violent. Another effectual means of restraint is a sheet across the trunk and arms, firmly secured at the sides of the bed. In most cases of delirium tremens this answers as well as the strait waistcoat, and has the advantage of being always available, but it also may slightly hamper the respiratory movements.

Food must be given in liquid form; beef tea, milk, and beaten-up eggs should form its chief part. The anorexia often renders it difficult to give nourishment, but the difficulty can generally be overcome by a little perseverance. It is well, in this as in other acute diseases, to commence the process of digestion of food before it is taken. Benger's Liq. Pancreaticus and Liq. Pepticus offer the readiest means for doing this, the former being used for milk, the latter for beef tea. Half a drachm of either may be added, and the food kept warm (not hot) for twenty minutes or half an hour, and then given. The process of digestion thus commenced goes on within the body.* If there is vomiting, peptonised enemata, well digested, must be given. The patient should not be without food for more than three hours; a due supply of nourishment is by far the most important element in treatment. Whether alcoholic stimulants should be given must depend on the state of the pulse. They are better withheld unless the pulse calls for them in language that is not only unequivocal, but is rather more decided than would be held a sufficient indication in an acute disease of other nature. Some stimulation may be obtained from ammonia and ether. Coffee has been recommended for the same purpose, and may be used after sleep has been obtained, or either caffeine or cocaine may be employed as a substitute. In all cases an aperient should be given at the outset, enough to open the bowels freely, but not so strong as to cause prostrating purgation.

It was formerly customary to administer sedatives freely from the first in the endeavour to procure the sleep that usually ends the acute disturbance. Bromides are commonly inadequate to compel sleep, although often useful as tranquillising agents; opium or morphia and chloral are the hypnotics usually given. In most severe cases efforts to produce sleep fail until near the time when the terminal sleep naturally comes. The insusceptibility to sedatives in the early stage is extraordinary; dose after dose may be given until a poisonous quantity is in the system, and still the patient may be sleepless, with increasing and perhaps alarming exhaustion. If the hypnotics are boldly pushed, they at last may act, and the patient may die from their influence. Anstie believed that when opiates fail to influence the brain and to induce sleep, they sometimes paralyse the heart. He mentioned a case in which, almost immediately after the administration of a second large dose of opium, the patient became ghastly pale, the pulse fluttered and then stopped, and in a few minutes the patient was dead.† Without doubt many cases have ended fatally in consequence of the idea that it is absolutely necessary to procure sleep at all risks. It is only in a very slight case that it is right to try to force sleep in the early stage. In all severe attacks it is wiser to postpone the attempt until the duration of the disease has increased the prospect of success—that is, until the third or fourth day of the attack. If a narcotic dose is given earlier (such as a quarter of a grain of morphia beneath the skin, or sixty grains of chloral by the mouth), it should not be repeated until sufficient time has elapsed to permit its action to pass off. It is better, however, in the early stage to be content with moderate doses, merely sufficient to favour trancause. *Acute mania* may thus come on, distinguished from delirium tremens by more persistent delusions and by the absence of tremor and of the restlessness of mind and body. Some of these cases run a shorter course than acute mania commonly does; sleep has a more pronounced ameliorating influence, and slight tremor may be present. Thus there are forms that seem to be intermediate between simple acute mania and delirium tremens. Chronic mental alteration is apt to be left behind for a time, but this, as we have seen, is not uncommon after delirium tremens in those who inherit a tendency to insanity. *Acute melancholia* also sometimes occurs from alcoholic excess, but does not present any special peculiarities. A peculiar form of recurring insanity, associated with, and excited by, a drinking tendency, has been termed *oinomania*. There is sometimes permanent moral deficiency, and there are periods in which the subject loses all moral sense, falls into various excesses, and may wander about and act in an absurd and often indecent manner. In some cases, during the intervals between the outbreaks, the patient exhibits no moral or intellectual defect.

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* This is a much better method than that commonly employed of carrying the process of artificial digestion further, and then stopping it by heat.

† Art. "Alcoholism," 'Reynolds' Syst. of Med.,' vol. ii, 2nd ed., p. 169.

CHRONIC ALCOHOLISM.

Various morbid states of the nervous system are slowly induced by habitual alcoholic excess. These differ from the acute disturbance in that they are often induced by habitual excess which never attains the degree necessary to cause actual drunkenness. The most common symptom is muscular tremor, seen chiefly in the hands, lips, and tongue. It is inconspicuous in the legs, although it may often be found there if looked for. It is a fine irregular tremor, and occurs only when the muscles are put in action by the will. It results from all kinds of excess, and may be as conspicuous in those who drink only beer as in spirit drinkers. It is generally greatest in the morning, and is less after some alcohol is taken. Sometimes there are also sudden starts of the limbs, especially during sleep. Insomnia is often troublesome; sleep, when it occurs, may be disturbed by distressing dreams, and visual hallucinations may distress the patient in the state between sleeping and waking, and may prevent sound sleep. To these symptoms some persistent mental changes are often added, irritability, restlessness, failure of memory. Other symptoms of disease outside the nervous system may co-exist, congestion of the face and conjunctiva, fulness of the eyelids, a tendency to skin eruptions on the face, a furred tongue, anorexia, and morning vomiting.

Besides these mental disorders, other definite maladies occur, subacute or chronic in character. The most important of these is multiple neuritis, a description of which was given in the first volume greater degree interstitial, and it may cause an isolated perineuritis (as of the sciatic nerve) to be combined with symmetrical polyneuritis.

It is an undecided question whether alcohol causes amblyopia and optic nerve atrophy, such as result from the use of tobacco. In many cases of tobacco amblyopia, there is a history of alcoholic excess, which has been thought to favour the occurrence of the affection, but its production by alcohol alone is not well established.

I have occasionally seen distinct slight optic neuritis in the subjects of chronic alcoholism, generally in association with headache and trifling mental changes. It is probable that in many of these cases there is chronic meningitis. Opacity and thickening of the arachroid and dura mater are met with after death in some cases, chiefly marked over the convexity of the brain, and are probably a direct effect of the alcohol. This, with some slight shrinking of the cortex, constitutes the most common post-mortem change in cases of long duration. Fatty degeneration of the walls of the small arteries is apparently more common in alcoholic subjects than in others. Minute foci of softening due to vascular disease, and called "encephalitis," have been met with in the cortex in rare cases, usually in association with considerable and long-standing mental changes.

Chronic alcoholism may aid in the production of many forms of definite insanity, but the only variety that can be certainly ascribed to this cause, acting alone, is chronic dementia—failure of memory, commonly progressive for a time, accompanied by defective power of judgment, and often by want of cleanliness and other indications of impaired moral sense. Mental weakness may be associated with defective articulation, and with recurring slight paralytic attacks, so as to constitute a group of symptoms bearing some resemblance to general paralysis of the insane, but differing in the non-progressive character of the disorder if alcohol is given up. Actual recovery is, however, rare, although the sufferers may live for many years. It is probable that, in such cases, there is chronic meningitis. The influence of alcohol in causing true general paralysis has been much discussed; the disease very seldom results from this cause acting alone, although intemperance may aid other influences in producing it.

The diagnosis of the effects of chronic alcoholism from the maladies with which they may be confounded has been sufficiently considered in the description of those diseases. The most common question is the distinction of alcoholic tremor from other forms, especially from the very similar tremor that is sometimes congenital, or, if not congenital, has existed from early life, and is often inherited. The history of the tremor sufficiently distinguishes the two affections.

The chief element in the treatment of chronic alcoholism is the change in the habits of the patient. Bitter tonics, such as bark, have been sometimes found of service, as in some measure satisfying the craving for drink. Tincture of capsicum has also been praised; ten-minim doses may be given before meals or when the craving for drink comes on.* Hydrochlorate of cocaine ($\frac{1}{10}$ to $\frac{1}{4}$ gr.) is also useful, and may be given after food, combined with half a minim of the alcoholic solution of nitro-glycerine ($\frac{1}{200}$ gr.), four minims of liquor strychniæ, and some liquid preparation of pepsin. The special derangements will generally subside when the cause ceases to act. The treatment that may be needed has been described in the account of these disorders. Strychnine and nux vomica are useful for the tremor. To lessen the insomnia and nervous restlessness, Marcet has recommended oxide of zinc, in two-grain doses, two or three times a day.

HYSTERIA.

The term "hysteria" is applied to a morbid state of the nervous system which is far more common in women than in men. The primary derangement is in the higher cerebral centres, but the functions of the lower centres in the brain, of the spinal cord, and of the sympathetic system, may be secondarily disordered. Hence the symptoms vary much, and the secondary manifestations may preponderate and obscure the primary disturbance. The name is derived from an erroneous idea that there is a special connection between the disease and disorders of the womb (ὑστέρα), which was even once thought to move about the body, and cause the local symptoms. The opinion that the disease is primarily and chiefly one of the cerebral functions is now all but universally held, and the present use of the word "hysteria" does not imply the theory involved in its derivation.

It should, however, be remembered that there is a difference between the popular and the medical use of the word. To the public, the name is chiefly associated with the simulation of symptoms, or at least with the idea that the patient could prevent the symptoms if she liked. But the malady is a real one, and to a large extent beyond the direct influence of the patient's will. It should also be remarked that the word is applied more widely to women than to men; the same conditions are often termed "hysteria" in the former, and "hypochondriasis" in the latter.

Etiology.—Both race and civilisation have probably more influence on the occurrence of hysteria than has been recognised or studied. It is almost unknown among barbarous races, and seems to be a product of the cerebral development that accompanies the process of civilisation. In certain stages of civilisation, peculiar forms of the disease have attained great development, as in Europe during the Middle Ages. Among races that have attained, apparently, an equal civilisation, hysteria reaches a higher degree in some than in others—in the French, for instance, and in the English; no doubt in consequence of the underlying differences in nervous constitution that are recognised in the expression "national temperament."*

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* See Ringer's 'Therapeutics,' 5th ed., 1876, p. 362.

Hysteria has been estimated to be twenty times as frequent in women as in men (Briquet), but the varying use of the word already mentioned renders the facts difficult to ascertain. Every form does occur in the male sex, and certain varieties are not unfrequent. The liability of females is probably determined by the character of their nervous system, and not by the possession of certain sexual organs. Some disposition to hysteria is inherent, if not in all women, at least in the vast majority. The affection is far more common in boys than in adult men; the differences in the nervous constitution of the two sexes are far less before than after puberty. Adult men who are the subjects of the disease often present mental characteristics resembling those of the female sex. The exact influence of age is also difficult to ascertain, because the special manifestations of the disease are the result of an underlying and antecedent morbid state, which develops so gradually that its commencement can seldom be determined. The frequency with which distinct symptoms commence in the several decades of life is shown in the following table, calculated from the figures given by Briquet and Landouzy:

Age . . .	—10	—20	—30	—40	—50	—60
Per cent. .	8	50	28	10	3	1

Thus in one half the cases the disease is first manifested in the second decade of life. Of these, a larger number begin between fifteen and twenty than between ten and fifteen. In nearly a third of the whole, the first manifestations occur between twenty and thirty, and a much smaller number in the first and fourth decades. The cases met with under ten generally commence after six, although symptoms of similar character have occasionally been met with at three, four, or five years of age (Barlow). The above statements are true chiefly of females. Regarding the age at onset in males, we can only say that the commencement is before puberty in a larger proportion of males than of females.

Heredity has an unquestionable influence, and may be direct, or a general neurotic tendency. One of the most severe cases of juvenile hysteria I have seen was in a girl of nine, both of whose parents had insane relatives. In another case, a girl had peculiar hysteroid fits, and both her mother and grandmother had had similar fits at the same age. Briquet found a history of hysteria in the parents of hysterical patients twelve times as frequently as in the parents of those who were not hysterical, but such a proportion is probably much larger than would be found to obtain in this country. It must be remembered that a tendency to hysteria finds favorable conditions for development in the injudicious moral training that is received by the children of an hysterical mother.*

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* The following account of the disease is based chiefly on its manifestations in the English race. Some features, that are merely alluded to here, have attracted great attention abroad. These have been, for the most part, fully described in the lectures of Charcot, and in the classical treatise of Richer, from which some illustrations are here reproduced.

The causes that directly determine the development of hysteria in an individual may be either physical or mental influences, or both. Of the two, the mental and moral influences are the more potent and the more frequent. They may both increase the predisposition and excite manifestations of the disease. The inherent predisposition, indeed, probably depends on the physiological preponderance of emotion in females, which is essential for the part they play in the continuance of the race, but which may be perverted either by congenital tendency or by injudicious training. License and indulgence in childhood beget self-indulgence in later life. When the disease has once developed, it is often greatly increased by injudicious management. The near relatives of the hysterical are often conspicuously deficient in judgment, and the little common sense they may possess is often rendered useless by their affection for the sufferers. In some cases, the symptoms develop without more exciting cause than the trifling annoyances of home life, or the change that the nervous system undergoes at the time of puberty. More frequently, however, there is some definite exciting influence—rarely intellectual exertion; in most cases it is an emotional disturbance. It may be a sudden alarm, which is especially effective in children. It may be merely the depressing emotions from which no life is exempt, trifling in themselves, but potent because unresisted. It may be some sudden and deep emotion, an unhappy love affair, a fall from luxury to the need for unaccustomed labour. The effect of the real causes may be merely seen in depression or irritability, often with failing health, and the outbreak may be immediately excited by some trivial disturbance, adequate only to turn the scale too delicately poised. Defective general health, though by no means essential for the development of the disease, is yet a common antecedent. Most patients are anæmic, easily fatigued, and often with functional disturbance of various organs.

Of the physical conditions that may influence the development of hysteria, disorders of the generative organs have always attracted most attention. Some morbid state of these organs is present in many cases, but the estimate that at least one half of the sufferers from hysteria are free from such disease (Jolly) is probably near the truth. Further, such disease, when it exists, is seldom the cause of the nervous affection. The morbid states of these organs that are met with are rarely grave; but they are occasionally such as cause frequent or continuous suffering, and thus may depress the nervous system.

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* See on this subject Dr. Russell Reynolds' address to the Sanitary Congress, 1887.

The rectification of such disturbance has, in a few instances, been followed by a marked improvement, but the symptoms of hysteria usually continue unmodified by the removal of the local disturbance, and the indirect influence of the treatment is often anything but beneficial. The effect of uterine disease or displacement which causes no obtrusive symptoms is more than doubtful.

The observations of Charcot have directed attention to the ovaries rather than to the uterus. Tenderness in the ovarian region is unquestionably frequent, but is not invariable, nor is it confined to the subjects of hysteria. It is usually a neuralgic affection, and not an actual local disease. It is very rare for there to be any adequate evidence even of congestion. When the generative organs are healthy, the menstrual period is attended, in most women, by some discomfort and depression, and it is natural that the symptoms of hysteria, if they exist, should be aggravated at these times. In such patients, moreover, menstruation is often irregular in consequence of the defective general health, and its disturbing influence is thereby increased. Severe cases of hysteria with much ovarian pain have been said to be cured by the removal of both ovaries; but too wide an inference may readily be drawn from the fact. The operation (of very questionable propriety) may remove a source of irritation, but it also involves a profound moral influence and prolonged physical rest.

Sexual excess is an occasional cause of hysteria in men, and, in its solitary form, still more frequently in boys, but its influence in women is probably not large. Continence has been supposed to be a cause of hysteria, but probably is so only when it suddenly succeeds habitual indulgence, and its influence is chiefly confined to the female sex.

Hysterical symptoms, especially convulsions, sometimes spread from one individual to another by sympathetic imitation,—"moral contagion," as it has been termed. Many singular outbreaks of this character have been recorded. The sufferers living together in a hospital, or workhouse, or school, have often been exposed to similar predisposing influences; and the materials in all were ready for explosion.

In a predisposed person the symptoms of hysteria may be excited or intensified by other diseases; nervous, general, and local maladies often determine the direction of more enduring hysterical disturbance. The symptoms which result from this union of disorders may be most perplexing. In typhoid fever in young girls, hysterical tenderness of the skin and spine may be present, and anæsthesia, pharyngeal constriction, rapid breathing, and even contracture may occur (Huchard). Tuberculosis is often attended with hysterical phenomena, which may simulate, or more often mask, the symptoms of tubercular meningitis. The secondary stage of syphilis, especially among prostitutes, is often attended by symptoms of hysteria, sometimes intense. Fournier believes that they result from a specific action of the virus on the nervous system, but the depressing effect of the temper, and an undue sensitiveness to annoyance, under which the trifling cares and vexations of life become grave troubles. Occasionally this deficiency of will-power is inconspicuous. Some patients with originally well-balanced minds, under the depressing influence of ill-health or care, may have their mental strength insidiously undermined, or a severe shock may disable the will, and the symptoms of hysteria may develop sometimes as disorders of subordinate functions. Occasionally, physical and mental depression may enslave to the lower forms of hysteria a mind which before despised the very name. Indeed, obtrusive contempt for hysteria is often a suspicious symptom, and may be due to dread of a tendency of which the sufferer is not quite unconscious, and a desire that no one should suspect the truth. The dread itself leads the mind to dwell on the nascent symptoms, and paves the way for their onset. But these cases are far less common than those in which the will has been deliberately allowed to fall into welcome servitude. Self-consciousness dominates, more or less completely, the patient's thoughts and even actions, and finds its expression in manner, glance, and tone. There may be obvious exaggeration in the description of sufferings, or an implied consciousness of much more than is expressed. The sympathy that is excited is a source of gratification to a patient whose sufferings often secure a relief from other annoyances which to her are greater, and the attention she receives is a new stimulus to her self-consciousness. The motives become stronger to yield to, than to resist, morbid tendencies, which are thus unconsciously cultivated. This defective will-power is sometimes associated with an imperfect perception of right and wrong, where desire is concerned, and the cultivation of symptoms, which is at first unconscious and involuntary, may then become conscious and intentional. Those that are at first merely unresisted may be afterwards welcomed, then invited, and at last actually induced or consciously simulated. Every stage in this gradation of development may be met with by itself, and sometimes, from the first, the symptoms are assumed. It must not, however, be inferred that all mimicry of disease is intentional simulation. The nervous system is dominated by idea and by fear, as well as by desire; the definite conception of a symptom may lead to its occurrence; and when idea and emotion are conjoined, and a symptom is not only conceived but either dreaded or desired, its occurrence is still more easy. The idea of a loss of power may render it impossible for the patient to will the movement; the conception of a muscular spasm may induce the contraction; and if a definite pain is thought of, before long it may be felt, without the symptoms being in any case intentionally induced. Medical inquiries and examinations often suggest to patients the definite ideas of symptoms, and the physician's knowledge of the natural association of symptoms may thus lead to their consistent grouping in a mimetic malady, even when there is not, and still more when there is, deliberate simulation. The pathogenic influence of idea is seen in all varieties of hysteria, and sometimes in singular isolation in children, in forms which Russell Reynolds has designated "ideal paralysis." The idea is most definite, and therefore most effective, when such symptoms have been actually observed in another person. Hence the spread of hysterical convulsions and other symptoms by imitative contagion. I have known two children in a family to suffer, one from a cerebral tumour, the other from startlingly similar symptoms of pure mimetic origin, evoked by the witnessed sufferings of the first.

The excess of emotion in the subjects of hysteria commonly finds free expression. Laughter and tears come readily, and these manifestations of emotion may occur in paroxysms on the most trivial excitant, and attend or constitute the slighter form of hysterical "fits." Certain other common symptoms of the disease are also natural results of profound emotion, and are morbid because spontaneous or too readily produced. One of these is the well-known "globus hystericus," a feeling of something suddenly closing the throat and stopping the breath. It is often described as a ball rising from the stomach to the throat; in other cases it is a mere sense of constriction or swelling referred to the fauces or pharynx; when intense there is probably actual spasm. An identical sensation may, from sudden alarm, occur in those who are not hysterical. It is a frequent precursor of an hysterical fit, but is also very common apart from any other paroxysmal symptom. The sensation, in its typical form, appears to be similar to that which immediately precedes many epileptic fits, and even some convulsive seizures from organic disease. It is often accompanied by a sudden sense of suffocation, for which there is no reason in the interference with breathing, and it appears to be the expression of a disturbance in the respiratory portion of the vagus centre, which is normally specially sensitive to emotion.

The same relation to emotion may be traced in many other symptoms of hysteria, which will be described in detail; such, for instance, as the disturbance of the heart's action, the limpid urine that may result from fear, and the muscular tremor of alarm, while many of the phenomena of hysteroid convulsions are but the frenzied manifestations of horror or rage.

Sensory Symptoms.—In hysteria all sensations, general and special, may be felt with abnormal acuteness; those that are in health unnoticed may give rise to distress, and the feelings are described in exaggerated language. The special senses may even be preternaturally acute; sounds may be heard which are inaudible to others, and the sense of smell is said sometimes to attain an acuity comparable only to that which it possesses in animals, so that persons have been distinguished by its means!

In these cases of increased sensitiveness painful sensations are readily produced and are often spontaneous, giving rise to the varied forms of hysterical tenderness and neuralgic pain, the locality of the two being often the same. The tenderness is frequently superficial, more pain being produced by a touch on the skin than by pressure on deeper structures; such superficial tenderness is common on the skin of the abdomen, thorax, and scalp, and sometimes affects one half of the body. There is often, in certain localities, deep-seated tenderness, and when considerable, the pain occasioned by pressure is peculiarly distressing, radiates to the chest or throat or head, and tends to cause the dyspnœa, sense of faintness, globus, and even in highly developed forms of hysteria, convulsive attacks. Hence these tender spots have been termed "hysterogenic" by Richer.

One of the most frequent seats for this tenderness is the ovarian region, and here it is commonly deep-seated. Its position is at the intersection of a line joining the antero-superior iliac spines with that which limits externally the hypogastric region; and Charcot believes that a tender body, which may, in a thin patient, be felt between the finger and the brim of the pelvis, is really the ovary, although in an opened body the ovary is usually found within the pelvis. It is doubtful whether the ovary is really felt, but it is probably tender, and so also are the adjacent parts. Weir Mitchell has observed extreme deep tenderness in this situation when the ovary could be felt, by vaginal examination, to be displaced downwards out of the usual position. Either ovarian region may be tender, the left more frequently or in greater degree. Occasionally, as Todd pointed out, there is extreme superficial tenderness in a circumscribed spot over the ovary. Tenderness in this region is, however, common in women who are not hysterical.

Another frequent seat of tenderness is the spine, especially the upper, middle, or lower dorsal region. The tenderness may be superficial, but more frequently it is deep-seated. Spontaneous pain, felt at more than one place, is often complained of, sometimes severe and burning in character, sometimes a dull aching which is compared to toothache. It is usually increased by exertion. Occasionally the whole of the vertebral column is tender and painful, and the pain seems to shoot up to the occiput. Sacral pain may be complained of, but sacral tenderness is rare.

Deep-seated tenderness in the left hypochondriac region is also frequent, and may even be greater there than in the ovarian region. Other occasional seats of tenderness, usually superficial, are the inframammary regions, and spots on the front of the abdomen and thorax, or on each side of the dorsal spine (Richer), which may be symmetrical on the two sides. Occasionally there is superficial tenderness (of the skin and sometimes of the muscles also) over the whole abdomen, and, simulating the tenderness of peritoneal inflammation, has received the absurd name of "false peritonitis." When there is general hyperæsthesia, the whole surface is "hysterogenic;" a prick on the forearm, for instance, may cause sharp pain, darting to the throat and causing globus.

The spontaneous pain, common in the left infra-mammary region, is apparently identical in character with that which is so common in the same situation in anæmia. Still more frequent, and very distressing, are the pains which occur in the head. The pain is sometimes frontal, temporal, or occipital, but much more frequently vertical, and because it is severe and occasionally described by patients as like a nail being driven in, it has received the name of the "clavus hystericus."

The hyperæsthesia of the special senses often occasions distress, which may be described as actual pain. Intolerance of light is very common, and is fostered by the dark blinds provided by sympathetic friends. Subjective sensations of various kinds may be complained of: noises in the ears, flashes of light or colour, or much less frequently sensations of taste or smell. Pricking or tingling sensations in the limbs, or vague feelings of numbness, are very common, and are often unilateral, affecting, for instance, one half of the tongue. Another sensation which is frequently described is that of cold water trickling down the spine. It is sometimes associated with a nervous shivering, analogous to that which is produced in healthy persons by a slight degree of cold combined with nervous excitement. Some joint may be the seat of spontaneous pain, attended, occasionally, with slight swelling such as may also occur in other parts, apparently from vaso-motor disturbance.

Lessened sensibility is very common, although often overlooked because the patient seldom complains of it, and she may, indeed, be unaware of its existence. Sometimes it occurs on the legs in association with motor weakness, but as an isolated symptom it usually affects part or the whole of one half of the body, constituting "hysterical hemianæsthesia," and may involve the special senses. When complete, a touch cannot be felt; a needle may be run into the skin without causing any sensation; neither cold nor heat can be perceived, and the skin may even be burned without pain. The loss extends up to the middle line, and may involve the mucous membrane of the conjunctiva, nose, mouth, vagina, and also the deeper structures, muscles, and bones. There are, however, certain anomalous features. Ovarian and other deep-seated tenderness continues on the affected side; reflex action is unchanged, the pupil still dilates when the skin is stimulated, and the fingers can still be used (*e. g.* for needlework) without the guidance of the eye. When incomplete, sensation may be lost to either pain or touch, rarely to temperature only. Often the sensation of touch is lost, and of pain only lessened; a prick may not be perceived, but faradism with a wire brush may be felt acutely. When partial in distribution, the arm suffers more than the leg, and the loss may be limited to the arm and cease abruptly at the shoulder or on the chest; the arm and leg may be affected, and not the trunk or face. The loss may reach the middle line in front, and stop far short of it behind. Loss of tactile sensibility may be more extensive than loss to pain. Occasionally only small areas are anæsthetic. The limbs are sometimes pale, and colder than those of the other side; a prick upon them may not bleed, but this is on the whole rare. Usually there is no difference in aspect or vascularity; and the mucous membranes are never paler. In health, pin-pricks in many places do not bleed. Hemianæsthesia is more common on the left side, and usually there is considerable ovarian tenderness on the affected side. Exceptionally I have found no ovarian tenderness, or tenderness on the opposite side.

The loss may come on spontaneously, or may follow a hysteroid fit. It may be present one day and gone the next; in one such case, a prick on the previously anæsthetic arm caused so much pain as to induce a hysteroid convulsion, and when this was over, the hemianæsthesia had returned, in typical and complete form. An increase may follow the testing of sensibility, and it is possible that an examination sometimes induces the anæsthesia. The area affected may vary from day to day, and may increase at menstruation.

The anæsthesia may also change from one side to the other without apparent cause, or such a "transfer" may be induced; a phenomenon discovered by Charcot, and much studied in France. The agents that cause the transfer may be (1) such as stimulate the skin and dilate the vessels, *e.g.* blisters, sinapisms, or faradism; (2) the application for half an hour of certain metals, especially gold, or of a large magnet or electro-magnet, which need not be actually in contact with the skin. Painting with collodion sometimes succeeds. The transfer is seldom lasting; after a few hours or a day the loss reverts to the original side. In the elaborate hysterics of France, sensitiveness to certain metals has been described (Burq, Dumontpallier, &c.), and it has even been asserted that patients may be cured by the internal administration of the metal to which they are externally sensitive! Mysterious and unknown forces have been invoked to explain the phenomena of "metallo-therapy," but they have received little confirmation elsewhere. Wood has been found as effective as gold, and so has a mental shock or the inhalation of nitrite of amyl.* "Metallic idiosyncrasies" have been practically unconfirmed, and the general evidence is conclusive in support of the opinion that the agents act through the mind of the patient—a theory in harmony with what we know of the pathology of the disease, and with many facts in its history, which show how much care is needed in drawing conclusions from the mysterious blending of psychical and nervous disturbance in its manifestations. If there is one lesson more clearly written in its history than another, it is that the more complex symptoms are best interpreted by the light of those that are more simple. The most powerful magnets that can be made, have not the slightest influence on the functions of the nervous system in a normal state.

As part of the hemianæsthesia, and capable of transfer with it, there

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* See a case recorded by Urbantschitsch, 'Arch. f. Ohrenheilk.,' Bd. xvi, p. 171.

(see p. 290). The glottis can be closed efficiently in coughing, except in very rare and extreme cases. Sometimes patients can sing well, although speech is whispered, and they have been known to speak in a loud voice during sleep. A scream may often be obtained by strong faradism applied either outside or inside the larynx. Hysterical aphonia may come on spontaneously, but is frequently excited by emotion; a circumstance which is not surprising when it is remembered that the larynx is the channel through which certain emotions are most readily manifested. It is also occasionally excited by laryngeal catarrh—the real loss of voice from cold persisting after the catarrh has ceased. The aphonia is not usually attended by any unpleasant sensation, but in one severe case the loss of voice was referred to a feeling "as if a bar of iron were laid across the chest." Speech often returns suddenly. The duration of the aphonia is extremely variable, and it is prone to relapse. One patient had been aphonic for ten years, with occasional intervals, during which the slightest fright would at once remove her voice.

Paralysis of the abductors of the vocal cords is an exceedingly rare consequence of hysteria. The symptoms are those described in the account of laryngeal paralysis. A few cases are on record, and I have seen one very striking instance, mentioned on p. 290. It is probable that this paralysis in hysteria has been sometimes mistaken for laryngeal spasm.

In rare cases of hysterical aphonia the tongue shares the laryngeal inaction, and loss of articulation is added to that of phonation, so that even whispered speech is lost, and the patient can express herself only by signs. In one girl, any sudden emotion would induce this state; it was accompanied by a sensation "as if the tongue were being twisted up." Simple aphonia may deepen into such absolute speechlessness. Sometimes there is a form of respiratory stammering. In a girl with hysteroid fits, one or two deep inspirations always preceded every attempt to speak, and sometimes interrupted a sentence or a word. During her fits she talked with perfect fluency. Rarely, actual stammering may be a part of severe hysterical disturbance.

Paralysis of the limbs is also common, and may assume the form of hemiplegia, of paraplegia, or of general loss of power. Certain forms of hysterical ataxy of movement may also be ranked in the same category. In this country, paraplegia is certainly more common than hemiplegia. As a rule, the onset in all forms is sudden, and often follows some emotion. The loss of power is usually moderate in degree at first, and gradually increases. The power which remains is put in action irregularly. Resistance to passive movement, for instance, is not sustained, but varies, at one moment being slight, at another considerable. If an attempt is made to execute a given movement, opponents of the acting muscles may be felt to contract. If the patient, for instance, attempts to extend the knee, the flexors of the knee may contract so as to hinder the movement. When clonus, it is probable that there is an alteration in the nutrition of the motor elements, although this may have arisen from functional disturbance.

Hemiplegia sometimes comes on suddenly, but more often slowly. It is at least three times as frequent on the left side as on the right. The loss of power is never complete; the leg is often more affected than the arm, and the face always escapes entirely,—important distinctions from hemiplegia of organic source. Loss of sensation usually accompanies the loss of power, but is more extensive in its distribution, affecting often the face and special senses. Reflex action from the skin is not lessened on the paralysed side, as it so often is in ordinary hemiplegia. The knee-jerk may be normal or it may be increased, sometimes on both sides. The spurious foot-clonus just described may sometimes be obtained on the affected side and not on the other. The jerky character of the muscular contraction in the arm may render its movement unsteady, when the leg is simply weak. Not rarely some persistent contracture of arm or leg accompanies the palsy.

Bilateral paralysis, a sort of double hysterical hemiplegia, is sometimes observed, although loss of sensation may be one-sided. In one case, a month after a whitlow on the finger, the patient, a girl, had some general pain, followed by inability to move the legs or arms, to swallow, or to speak. The fingers became strongly flexed, with the thumb thrust between the two middle fingers. In this state she lay for a month. Then the fingers became relaxed, the arms were moved a little, and at times a word or two was jerked out with difficulty. The influence of faradism restored her speech in a few days, and in a fortnight she had regained full power of limb.

Disorder or ataxy of movement occurs in the hysterical in many forms, and may exist alone or accompany loss of power. One form, well described by Briquet, in which movements are steady under the guidance of the eye, but become irregular as soon as this guidance is withdrawn, is supposed to be due to "muscular anæsthesia." But this pathological condition has been invoked without sufficient reason to explain other forms of inco-ordination. The varying force of muscular contraction, which may be often recognised in the resistance to passive movement, imparts a jerky unsteadiness to voluntary movements. Sometimes these are steady enough while the patient is in bed, but on standing she at once sways, first to one side and then to the other. Or, without any loss of cutaneous or muscular sensibility, the patient may be unable to stand with the eyes closed, although perfectly steady when the eyes are open, the effect of closure being greater than is commonly seen in true ataxy apart from impairment of sensation. In other cases there is a tendency to fall backwards.

In the functions of the cranial nerves, the only other paralytic affection of hysterical origin is the peculiar form of simulated ptosis described at p. 201. Ordinary facial palsy, in these patients, has been described as hysterical, without any reason. Inaction of the diaphragm may closely simulate a true paralysis. The diaphragm naturally acts less in women than in men, and may take little or no share in deep voluntary breathing, which is chiefly effected by the superior thoracic muscles. In proportion as respiration is influenced by the will, the diaphragm is inactive. The effect of observation on an hysterical patient may be to render the breathing almost wholly volitional, and to throw the diaphragm out of action. Repeated examination, and the diversion of the patient's attention, will generally reveal the absence of real paralysis.

Spasmodic Affections.—The Protean character of hysterical disorders is nowhere more conspicuous than in the varied forms of spasmodic affection to which the disease may give rise. They may be persistent or paroxysmal: the former class includes the forms of tonic spasm, or contracture, and the persistent varieties of clonic spasm; the latter comprehends the various degrees and forms of hysterical convulsion.

The term "contracture" is applied to the condition in which the muscles become rigid in tonic spasm, fixing a limb or limbs in a certain posture, for a few minutes or for a longer time. The contracture commonly succeeds a hysteroid fit. Occasionally it is excited by some local injury or local pain, as that of neuralgia or rheumatism; less commonly it comes on spontaneously. In most cases it is uniform while it lasts, but occasionally the muscular spasm may be felt to vary in intensity from time to time. It is usually greatest when attempts are made to overcome it; the varying resistance of the muscles, then felt, is characteristic, and of much diagnostic importance. The contracture usually persists during ordinary sleep, and is only relaxed by the deepest chloroform narcosis. In extremely rare cases, in which it has existed unchanged for years, structural alterations appear to take place in the muscles; the contracture leads to organic shortening, and can no longer be removed even by chloroform (Charcot).

One form of contracture is that of the muscles of mastication, causing hysterical trismus, in which the teeth cannot be separated for more than a quarter of an inch. Usually succeeding a fit, it often continues until another convulsion, and then is gone. Sometimes it comes on spontaneously. It seldom lasts more than a few days, but is prone to recur. One patient, for instance, had an attack, lasting a quarter of an hour, three or four times a day for several weeks. Very rarely there is transient contracture in the depressors of the jaw, keeping the mouth wide open.

Contracture in the limbs may involve the arm, alone or with the leg on the same side, or the two legs may be affected, or it may be general. The arm is the most frequent seat. It is always rigid in flexion; the elbow is bent at a right angle, or even still more flexed; the wrist is flexed; the fingers are sometimes flexed at all joints, as in the "late rigidity" of hemiplegia, with the thumb beneath them or thrust between the first and second fingers. The contracture in the fingers is not lessened when the wrist is passively flexed, as it is in the late rigidity of hemiplegia. Sometimes the digits are flexed at the metacarpo-phalangeal, and extended at the distal, joints, from the preponderant contracture of the interossei; the posture then resembles tetany. The forearm may be pronated or supinated. Contracture in the arm may follow a fit, and may be transient or continue till another fit. Injury is a very common cause of contracture; from this, it usually comes on gradually and slowly; as long as it is local, it is often thought to be due to a neuritis; but if, as is sometimes the case, it spreads beyond the arm, involving the leg on the same side, or becoming universal, its nature is clear. As an instance of this cause may be mentioned the case of a girl of sixteen, who let a hot iron fall on her left wrist, burning it slightly. Immediately she felt a pain in the thumb and lost feeling in it. A week later, and in the course of a few days, first the thumb and then the fingers became flexed and rigid, one after another. Then the elbow became partially flexed, and soon afterwards the foot was strongly extended and the toes bent downwards. In the hand and foot there was fine tremor during observation. Sensibility was lost in the whole arm but nowhere else. Ovarian tenderness existed on the same side, and the contracture could be partially removed by firm pressure there, or by faradisation of the limbs, but quickly returned. Under moral treatment, however, it gradually passed away, and at the end of two months no trace of it remained.

Hemiplegic contracture, such as the above case illustrates, is not very rare. Some loss of power is usually associated with it, and, as in simple paralysis, the face is always free from rigidity. Anæsthesia is rarely absent on the hemispastic side, and the loss of sensibility may be complete and involve the face and trunk as well as the limbs. Sometimes, as in the above case, the loss of sensibility is partial and is confined to the most contractured limb, or there may be absolute loss in it and a slighter loss over the whole of that side.

The contrast, in the case just described, between the form of spasm in the arm and leg, is common to almost all cases of hysterical contracture; the arm is fixed in flexion, the leg in extension, and the latter may be transformed into a rigid bar, fixed to the pelvis by unyielding spasm, with the heel so drawn up that the dorsum of the foot is in a line with the front of the leg. The foot is commonly inverted and the toes flexed. Partial contracture affects most the extremity of the limb. In these slighter cases the toes may be over-extended, in spite of the heel being raised. In hysterical contracture with extension of the ankle, the foot-clonus can often be obtained, as Charcot has shown. It is analogous to the physiological clonus which occurs in most persons after standing for some time on tiptoe.

Paraplegic contracture, affecting both legs, is less common. The position of the legs is that already described, rigid extension; the flexor spasm, occasionally seen in organic disease, is scarcely ever be observed to rise in consequence of the relaxation of the diaphragm. The peculiar affection termed "tetany" (p. 698) sometimes occurs in the subjects of hysteria, but it is usually trifling in degree, and is limited to the hands. In some cases of hysterical tetanoid contracture the fingers are extended and separated by the spasm.

Clonic Spasm.—Tremor is very common in the hysterical, especially as an accompaniment of paralysis and contracture. It is rarely constant, but is usually evoked by movement and excitement. Frequently it does not attend the commencement of movement, but if the muscular action in the arms or legs is maintained for a few moments, the limb is agitated by fine quick tremor, varying in degree and in time, always increased, and sometimes distinctly induced, by attention, and when the mind is diverted the tremor may cease. When the legs are the seat of contracture, the shaking is often caused by attempts to move them, or even by the manipulation of a medical examination. In all these respects it differs from the tremor of paralysis agitans, while the fineness of the tremor, and the absence of actual inco-ordination, distinguish it from that which is characteristic of disseminated sclerosis, although in this disease there is occasionally tremor very much like that of hysteria.

Such tremor, widening in range into what deserves the name clonic spasm, is occasionally paroxysmal, affecting the head or limbs, produced by emotion, and often to some extent under voluntary control. A young unmarried man, of highly nervous temperament, was liable to outbursts of hysteria, of which such clonic spasm was an invariable part. Under the excitement of a medical examination, for instance, his head began to shake violently. He said that the shaking in the head distressed him peculiarly, and that he could by an effort send the shaking into his leg, where it was much more bearable. Presently the right leg quivered in quick spasm, approaching in rapidity the foot-clonus, and the head was still. Similar spasm, general in distribution, is a conspicuous feature of many of the hysteroid convulsions presently to be described.

Local and persistent clonic spasm is a most troublesome but happily a rare symptom of hysteria. An example of it was presented by a girl who had two attacks of such spasm in the left pectoralis muscle, jerking the shoulder forwards with great force. The spasm was regular in time, but irregular in force, and during the waking hours it never ceased. Each attack lasted for several months. A circular blister around the arm arrested the first, but had no effect upon the second, which resisted all treatment until a journey to the south of Europe cured it. A similar spasm may sometimes be seen in the muscles of the neck, causing a sort of spurious torticollis. It is usually bilateral, jerking the head backwards or forwards; the movement is slight in range and quick in time, and thus differs from the more extensive and deliberate movements presently to be described.

Certain forms of widely distributed clonic and irregular spasmodic movements have been called "hysterical chorea," but they differ much in their characters, and to most of them the term chorea can only be applied when used as a generic designation for all persistent involuntary movements. It is not uncommon, however, for witnessed chorea to be reproduced by the imitative tendency of hysteria; the mimetic malady may closely resemble its original, the movements being varied and irregular, but much more often the muscular contractions are more sudden and shock-like than in true chorea, resembling in this the "electric" form. The imitative form is usually transient, and quickly vanishes when the patient is withdrawn from the influence of example. Occasionally, similar forms of choreoid affection come on in these patients apart from imitation, and these are frequently most obstinate affections, lasting sometimes for years. Often the movements, in the hands especially, have a rhythmical character. There may be, for instance, quick, regular, flexor movements of the fingers of each hand, all joints being bent. The movement is always increased by notice, and as it lessens in degree it may occur only when the attention is directed to it. (See also p. 610.)

Rhythmical movements of more complex, and therefore more deliberate character, and of wider range, have also been included under the term "hysterical chorea" by the French (after Germain Sée, &c.), and constitute the disease long known as "chorea major" by the Germans. The movements are wide in range and regular in sequence, and consist of alternating contractions in opposing muscles, especially the flexors and extensors of limbs and trunk; they cause an oscillatory motion as regular as the movement of a pendulum, ceasing only during sleep, and lasting for days, weeks, or months. The head may be thus moved from side to side, backwards or forwards, the jaws up and down; or, less commonly, the tongue may be alternately protruded and withdrawn, the eyelids rapidly closed and opened. Much more frequently one limb is thus moved, or the arm and leg on one side, with or without the trunk, very rarely all four limbs. Briquet has related a case in which the leg was flexed until the foot touched the forehead and again extended, in regular sequence for more than a year, in spite of treatment, until the movements were arrested by violent emotion.*

Rhythmical, co-ordinated spasm of these forms is analogous, as Charcot has pointed out, to that which occurs, in paroxysmal form, in many hysteroid convulsions. In all cases it is probably pathognomonic of hysteria, and may occur at the onset of other forms of hysteroid affection. A girl of seventeen, while at supper, became suddenly faint, and immediately the right arm became the seat of rhythmical flexion and extension movements; in half an hour the leg was affected in the same way, and then the other limbs, and for five or six hours these movements went on without any spasm in the trunk. They ceased when sleep was obtained by a hypodermic injection of morphia, and there was no return, but the patient woke to pass into a state of contracture and paresis which lasted for several months.

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* A striking example of unilateral spasm of this character is related by Charcot, 'Brit. Med. Journ.,' 1878, i, p. 221.

Convulsive Attacks.—Among the paroxysmal symptoms must be included the most frequent of all hysterical phenomena, the globus hystericus, and the paroxysmal manifestations of emotion, which have been already described. Just as emotion is naturally expressed by muscular actions, in the "jumping for joy" of a happy child, the stamping of rage, or the wrung hands of distress, so the violent emotional discharge of hysteria may be associated, in more severe cases, with violent movements of the limbs, purposive in aspect, but purposeless in aim. The arms and legs are dashed about with violence, the head is thrown from side to side, the back may be arched by contractions of the spinal muscles. This is the mildest form of hysterical convulsion, unaccompanied by loss of consciousness—the "hysteria minor" of the French.

In still further degrees of intensity, the convulsive phenomena become much more complex and severe. The attacks occur with less dependence on exciting emotion, and present more distinct alteration in the mental state. These are the attacks of "hysteria major" of the French, and from a frequent resemblance to epileptic attacks, they have long been known by some compound name, and especially, through the influence of Charcot, as "hystero-epilepsy." The term, however, when applied to phenomena of pure hysteria, is inaccurate, and it is better to call these seizures "hysteroid"[*] or hysteria major.

In the severe attacks, rigid fixation of the trunk and limbs, often opisthotonic, alternates with wild movements, in which the limbs are thrown about with great force and rapidity, the arms strike out, the legs kick, the head is dashed from side to side. These phenomena may be varied by quiet intervals, often attended by hallucinations or delirium. Consciousness may be apparently lost, or manifestly perverted, and, as a rule, the patient retains no recollection of the fit. In the most severe and elaborate forms of attacks, such as occur especially among the French, certain stages may be distinguished, of which Richer has given careful descriptions and striking illustrations.[†] The attack is often preceded by a period of mental disturbance, with hallucinations. The onset is attended with sudden loss of consciousness, and general tonic spasm, followed by clonic spasm. These constitute the first or "epileptic" stage. Then sometimes, after a brief interval of coma, the second stage of co-ordinated spasm, or "*grands mouvements*," comes on, opisthotonos, bounding movements of extreme violence, succeeded by the third stage of mental and emotional disturbance, in which the patient talks deliriously, with manifestations

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[*] A term suggested by Sir W. Roberts, which, although not perfect, is less inexact.

[†] 'Études cliniques sur l'Hystéro-épilepsie,' a work in which careful observation, exact description, and artistic skill are combined in a remarkable degree.

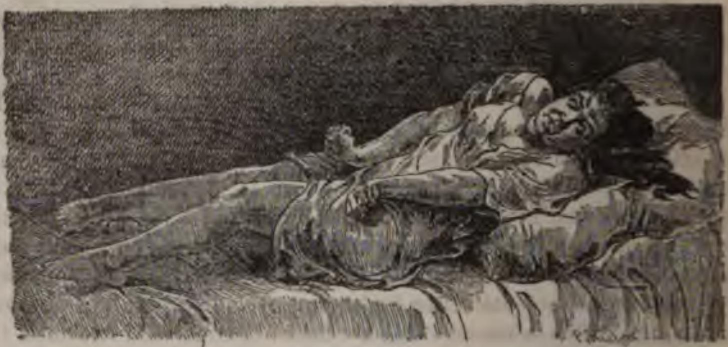


Fig. 175.—"Hysteria major." First or epileptoid period. Phase of tonic immobility or tetanism. (Richer, Pl. II.)

Fig. 176.

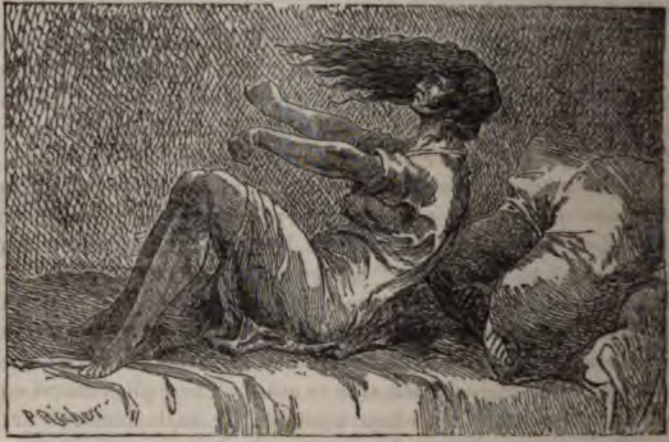
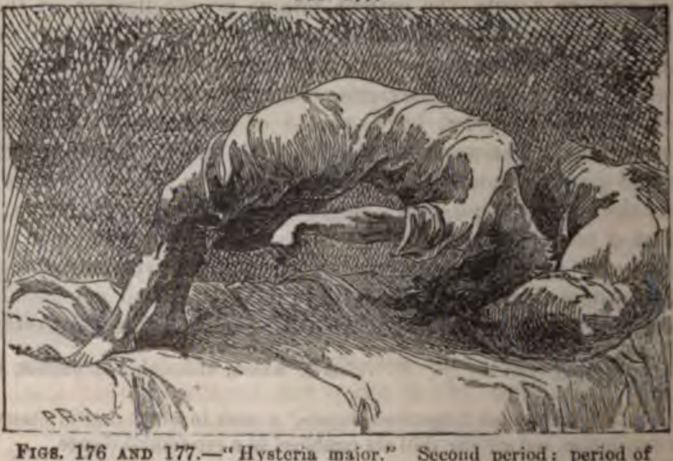


Fig. 177.



Figs. 176 and 177.—"Hysteria major." Second period: period of clownism. Fig. 176. Phase of violent movements. Fig. 177. Phase of contortions, "*arc en cercle.*" (Richer, Pl. III.)

FIG. 178.

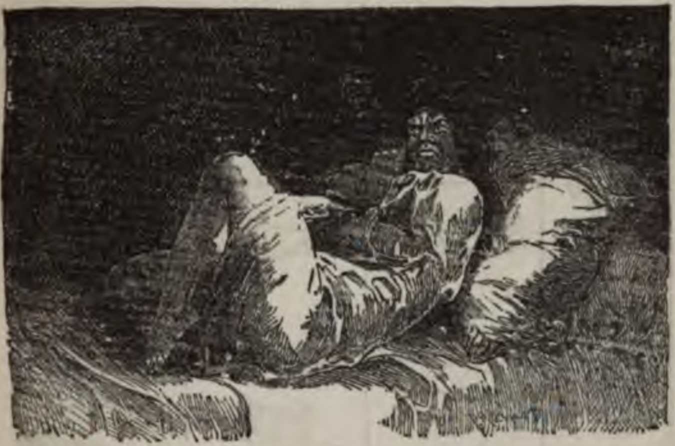
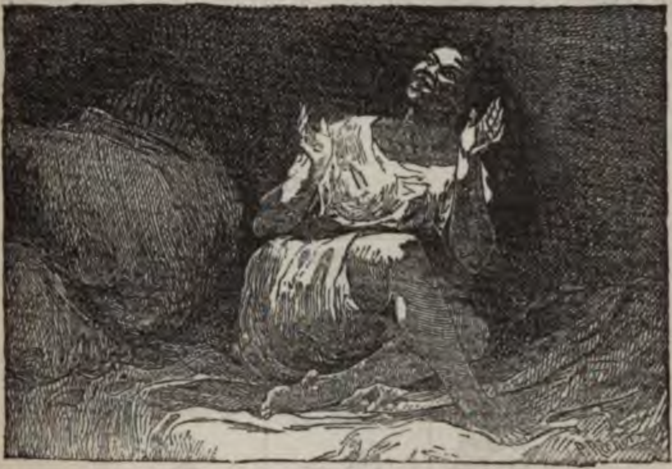


FIG. 179.



FIGS. 178 AND 179.—"Hysteria major." Third period: period of emotional attitudes. Fig. 178. Sad phase. Fig. 179. Gay phase. (Richer, Pl. IV.)

of joy, anger, or erotism. Tenderness of the ovarian region is almost always present, and by pressure there a fit of the above description may be at any time induced, or may at any period of the attack be at once arrested.

The kindness of M. Richer permits me to reproduce some of his illustrations. The figures, although failing to do justice to his remarkable etchings, yet afford a graphic illustration, and useful epitome, of the phenomena of the severe attacks as observed at the Salpêtrière.

Fig. 180.

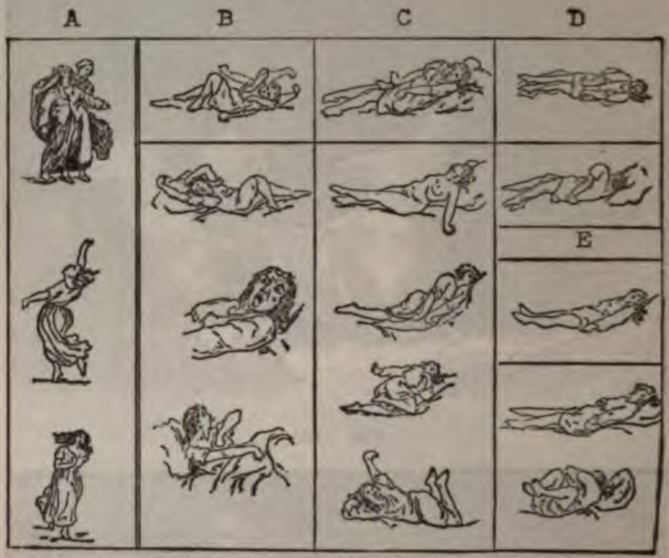


Fig. 181.

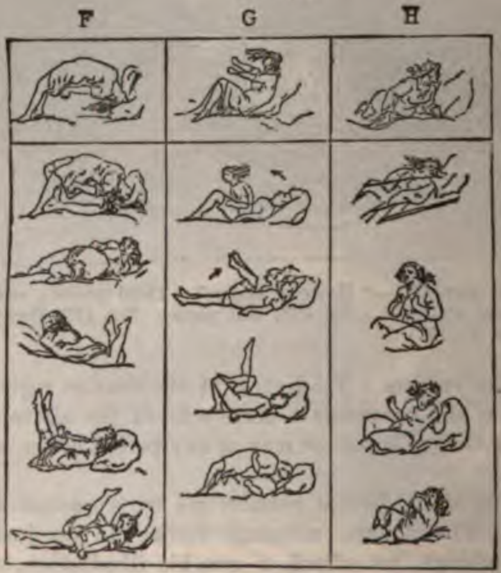
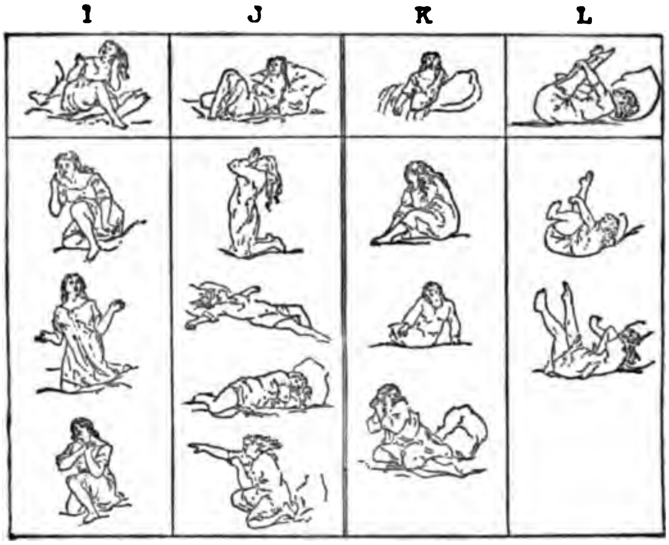


Fig. 182.



Figs. 180, 181, and 182.—" Hysteria major." Synopsis of the features and varieties of the attack of Hysteria major. (Richer, Pl. V.)

The highest figure of each vertical series (above the horizontal line) represents the commonest feature of each successive stage, indicated by the letters; while the figures below in each column represent frequent varieties of the common type of that stage.

Prodromata			A
Tonic phase	Violent tonic movements		B
	Tonic immobility		C
First period	Clonic phase		D
	Phase of resolution		E
Second period of clownism	Phase of contortions		F
	Phase of violent movements	Rhythmical	G
		Disordered	H
Third period of emotional attitudes	Gay		I
	Sad		J
Fourth or delirious period	Delirium, zoopsia		K
	Generalised contractures		L

In this country the attacks rarely correspond closely to this description; they present similar phenomena, but in less regular sequence, and often in isolated form. The initial stage seldom presents any close resemblance to an epileptic fit. There is often initial tonic rigidity, and this is sometimes followed by a form of clonic spasm, succeeded by the co-ordinated movements, but the clonic spasm differs from that which occurs in epilepsy, and not unfrequently the patient passes at once into the violent co-ordinated movements, in which paroxysms of tonic and clonic spasm occur from time to time.

It may be well to consider separately the various phenomena of these attacks as they are met with in this country. Their occurrence is often distinctly aided by, or due to, emotional disturbance,—prolonged annoyance or sudden alarm. The premonitory mental disturbance with hallucinations is never met with, but the attacks are often preceded for some hours by headache or some general dysæsthesia. Immediate warnings are not uncommon, especially a sense of illness, the throat *globus*, giddiness, palpitation of the heart, or some sensation commencing in *both* feet and ascending to the head. These warnings are common in pure hysteroid fits, but it is important to remember that these attacks sometimes occur as sequelæ to epileptic fits of slight or moderate severity (see pp. 743, 747), the warning of which must not be mistaken for the warning of the hysteroid attack. In the simple hysteroid attack there may or may not be pallor before the attack comes on. The patient may fall, occasionally with some force, but never suffers the injuries so common in epilepsy from falls upon dangerous objects or on the fire. Very often the fall is gradual, a sliding to the ground rather than a fall. When there is initial tonic spasm, the limbs are usually rigid in extension, the toes pointed downwards. The arms may lie by the side of the body, or be extended at right angles to the trunk in the attitude designated "cruciform" by Charcot. The fingers are usually flexed at all joints, the fists being clenched. There is never, in this stage, the "interosseal position"—the flexion of the metacarpo-phalangeal and extension of the other phalangeal joints, which results from preponderant spasm in the interossei, and is so common in epilepsy and in some hysterical contractures. During the existence of the tonic stage, the foot-clonus can often be obtained, as Charcot has pointed out.

The opisthotonic spasm is one of the most characteristic features of hysteroid convulsion. It is the *arc en cercle* of French writers (Fig. 177). It occurs especially during the stage of co-ordinated movements, rarely at the commencement of the fit, although the initial tonic spasm may pass into it. There may be only slight arching of the spine, or the contraction of the extensor muscles may be so severe that the patient rests on the back of the head and the heels, and, in extreme cases, the trunk is pushed up by the feet, and the neck so much bent backwards that the vertex, or even the forehead, is the anterior point of support, and it seems as if the neck would be broken. It rarely continues for long, but the patient from time to time may sit up, and then bound backwards into the rigid arch. Opisthotonos may also occur as the patient lies on the side, the *arc en cercle latérale* (Fig. 181).

The clonic spasm varies very much in its character. It never resembles precisely that of epilepsy, in which the movements are violent and shock-like in their character, and cease by gradually becoming less frequent, not less strong. In hysteria the spasm is rarely shock-like, and always maintains the same frequency until it suddenly ceases. It is usually very quick in time. Occasionally the conception of a universal clonus, like that of the foot, but affecting whole limbs, conveys the best idea of its character. The fit may commence with this, without any initial tonic spasm. In the rare cases in which the spasm is shock-like, the shocks are infrequent and deliberate, and go on for a long time. Much more frequently the spasm is rather coarse tremor or quivering. The movements may affect the hands and feet only, so that these are struck against the ground with great rapidity. The orbicularis palpebrarum is often alone affected, causing quivering of the eyelids, or quick opening and shutting movements. The tongue is never bitten in the clonic spasm as it is in epilepsy, although, very rarely, it may be bitten accidentally in the fall. Hysterical patients, however, often bite their lips.

The co-ordinated movements which constitute so large a part of the attack are, for the most part, wild, irregular "fighting" or struggling movements, in which the legs, feet, and head are thrown about with great violence. The movements are usually without serial order, but occasionally certain movements are repeated in a rhythmical manner. The head may be moved from side to side; there may be regular flexion or extension movements of the legs (sometimes propelling the patient head first along the floor), and still more frequently a similar rhythmical movement in the arms. These co-ordinated movements are conspicuously increased in force by attempts to restrain them; the more force is used the more is needed; and their violence is often extreme. Accompanying them, there is often delirious mental disturbance, most conspicuous during intervals of comparative rest or of alternating tonic spasm. Sometimes the patient talks in an unnatural manner, manifesting some hallucination. One girl, for instance, thought her long black hair, which had been flying about her head, was seaweed. Usually there is more or less emotional disturbance, especially manifestations of terror, which may increase to maniacal frenzy (Fig. 178), and culminate in a paroxysm of convulsion. An ecstatic stage, with passionate attitudes, as depicted by Richer in Fig. 179, and also indicated in its varieties in Fig. 182 J, is seldom met with here, and it is equally rare for there to be a stage of fixed contracture, as in Fig. 180 B. There is often a strange tendency to bite in a curiously animal manner, and occasionally noises of animals are imitated (therio-mimicry).* The patient will make a sudden gnash at the hand of an attendant, and if not prevented, may inflict serious bites on her own fingers. In a prolonged seizure there are often intervals of tranquility, in which the patient may seem well, but, by slight peculiarities in manner, those who know her are aware that "she is not yet herself," and presently the spasm suddenly recommences.

The eyelids in hysterical attacks are usually closed, except during the delirious phase, when the eyeballs often converge strongly from time to time. Reflex action from the conjunctiva is usually lessened. General sensation is distinctly diminished; a pin may be run into the skin without causing any evidence of pain. In rare cases the attacks like other nervous diseases in females, may be accompanied with symptoms of hysteria, without the association proving any essential relation. Sometimes, however, pronounced mental aberration seems distinctly to grow out of the slighter psychical disturbance of hysteria, and the process may be apparently similar to that which leads to the more grave corporeal symptoms. Just as a mind unfixed by emotion may become set, in its unbalanced state, by a fixed idea of motor inability, and the latter may progress to extensive paralysis or contracture, so the intellect may become the prey of such an idea as must be regarded as an insane delusion, under the influence of which the mental powers may fail further. An emotional girl, for instance, with frequent globus, became possessed with the idea that all persons whom she met were making faces at her, and then she manifested timidity, infantile in its degree, so that she dared not go out of the house alone, and became irritable and depressed, with a vacant look, and such loss of memory as to constitute partial dementia. Such cases may be most various in their form, and the ultimate symptoms may resemble those of simple insanity, from which they are to be separated, if at all, only by the conditions of their origin.

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* See 'Epilepsy and other Chronic Convulsive Disorders,' p. 140.

There is another important relation of hysteria to insanity. The later stage of a developed hysterical fit is a paroxysm of mental disturbance of maniacal violence, in which chaotic storms of emotional frenzy alternate with hallucinations and delirium. The patient is, for a few minutes, wildly insane. Just as some of the motor spasm of the hysteroid convulsion may continue, or may occur alone, as persistent contracture, so some elements in the paroxysmal mental disturbance, hallucination or delirium, may occur alone. In an hysterical child, whose case I have described at length elsewhere,* apart from the delusions that accompanied the convulsive seizures, there were occasional periods of spiteful mischievousness, wholly foreign to her habitual character, and also periods of a semi-demented state, lasting for days or weeks. The subjects of such mental disturbance may threaten or attempt suicide, apparently with every intention of success. In another patient,† a girl of twenty-six, violent delirium accompanied each hysteroid convulsion, and after a time paroxysms of mental disturbance occurred without any preceding fit. After an interval of improvement, a condition came on which rendered her confinement in an asylum necessary. She recovered, but relapsed, in consequence of a mental shock, and passed into a very unpromising state, with most degraded habits, but ultimately became well.

Visceral and Vaso-motor Symptoms.—The digestive, respiratory, and circulatory systems are liable to be deranged in hysteria. Some of the resulting disorders are very remarkable in character, and indicate how profound may be the secondary influence of the disease on subordinate functions.

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* 'Epilepsy,' &c., p. 157.

† Loc. cit., p. 172.

days. In some of these cases, forced feeding by the mouth, or even by the rectum, may give rise to such severe convulsions that the attempt has to be relinquished. The patients lose weight, and become emaciated and feeble in extreme degree; the temperature may be raised, the tongue becomes dry and brown; they may pass into a condition of stupor, and not only appear in danger of immediate death, but may actually sink from exhaustion. In most cases, however, when this alarming state is reached, a little food is again taken, and the patient slowly revives. The danger is greater in cases of prostration from long-continued vomiting than from simple refusal of food, since forced feeding is less effectual. One patient died on the eighty-second day of the vomiting, in spite of the use of the stomach-tube.*

Dyspepsia, in every form and degree, is common in the hysterical, and the nerve disturbance thereby excited may profoundly intensify other symptoms. Sensations of "sinking," palpitation, breathlessness, flushing of the face, pains in the back, which often attend dyspepsia, may reach an extreme degree in the subjects of hysteria. Flatulence, gastric and intestinal, is very common, and gives rise to much discomfort and to varied sounds, some of which are apparently the effect of an involuntary contraction in the muscles of the abdominal wall. Complicating and intensifying these troubles, there is often constipation, which, so common in women, is peculiarly obstinate in the hysterical. The bowels, left alone, may act only once a week or even once a month. Whether this results from a nervous inhibition of the intestinal wall, or from mere indisposition to aid the bowels by timely attention, is difficult to say. It is certain that the muscular action of the intestines may be gravely deranged in severe hysteria. A case is recorded by Briquet, in which liquids injected into the rectum were vomited by the mouth. Even tincture of litmus was vomited twelve minutes after its injection into the rectum, the patient being watched by doctors and nurses the whole time. That there is a pathological inactivity of the bowel is probable from the large amount of purgative which is needed. Occasionally there is a peculiar sensitiveness to the action of the bowels, so that the passage of a stool, especially if relaxed, may cause sensations of faintness, and even actual syncope.

Retention of urine is common in the hysterical, but incontinence is almost, if not quite, unknown. Calls to micturate are often annoyingly frequent, but this usually depends on the character of the urine, which is often abundant, pale, and of low specific gravity, and seems to irritate the bladder much more than that which is less dilute. Such urine is probably due to dilatation of the renal vessels. It is secreted in most persons under the influence of emotion, and may be almost constant in the persistent emotional state of the hysterical. In rare cases the secretion is changed in the opposite way. Instead may occur apparently without cause. There is undue consciousness of the action of the heart, normal or irregular, which is described as "palpitation." It is often a source of much distress, and may be accompanied by cardiac pain and giddiness, by sensations of sinking, of dyspnœa, and of faintness, by extreme pallor, and even by actual syncope. Sometimes the deranged innervation is shown, not only by an habitual frequency of 120 or 130 beats per minute, but by a reversal of the usual effect of posture on the pulse. The attacks of pain, with pallor, faintness, and dyspnœa, may closely counterfeit genuine anginal seizures, especially when the pain, as is sometimes the case, radiates to the left arm; if the symptoms of angina pectoris are known, dread of it augments the distress. In special conditions of depressed brain function, the heart's action becomes infrequent, especially in some of the cases of spontaneous trance.

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* Guyot, 'Gaz. méd. de Paris,' 1882, p. 206.

Disturbances in the peripheral vaso-motor system may be associated with the perturbed action of the heart, or occur independently. Flushing of the face, sometimes local in distribution, is exceedingly common. It occurs spontaneously or on slight emotion, and from the misconstruction to which it gives rise is a source of great annoyance to the patient. Flushing and pallor may alternate, or the face be red and burning while the feet and hands are cold. Flushing of the feet, with a sensation of burning heat, often alternates with cold, and much more rarely there is persistent fulness of the vessels of both legs. Weir Mitchell has recorded an extraordinary case in which at times, through emotion or spontaneously, a paralysis of the abdominal vessels seemed to concur with vascular spasm elsewhere, so that the flat abdomen of a thin, spare widow would attain, in the course of a few hours, a size corresponding to the sixth month of pregnancy, and be turgid and pulsating, while the rest of the body was pale and bloodless, the pulse at the wrist being a mere thread, and faintness ensued if the patient sat up. The state would subside in the course of a few days, but it was the source of infinite annoyance to the patient.

Local perspiration is a rare symptom. In one case the hands and feet were the seat of profuse sweating, which alternated with attacks of lethargy, transient double amaurosis, paraplegia, and anæsthesia (Siredey).

Vaso-motor spasm seems sometimes to accompany hemianæsthesia, so that pricks do not bleed, but, as we have seen, it is no necessary concomitant; in complete hemianæsthesia the vascularity of the skin may be normal, and pricks may bleed readily.

Occasionally local swellings occur, especially about the hands or feet, or about joints, sometimes in the seats of neuralgic pain. They are due apparently to the effusion of serum into the cellular tissue in consequence of vaso-motor disturbance, and often occur near the time of menstruation.

It is probable that, in rare cases, the vaso-motor disturbance may married state, and which not rarely constitute the greatest trials life can bring. Hence severe hysteria often persists to middle life and even beyond it, in some cases to cease, in others to be intensified, at the climacteric period, and occasionally to continue until old age.

The course of the disease is always varied. The infinite diversity of symptoms which different patients present, may be almost paralleled in the history of individual cases; palsies, motor and sensory, contractions and convulsions, dyspnœa and dysphagia, anorexia and aphonia, faintings and vomitings, may succeed one another in apparently inexhaustible variety.

In what proportion of cases a practical recovery takes place cannot be determined. As a rule, however long the symptoms have lasted, they remain disorders of function only, and their complete disappearance is always possible. To this an exception must apparently be made in the case of persistent contractures, which, as already stated, after lasting for many years, may be attended with structural changes in the spinal cord; such cases are, however, excessively rare.

Recovery from hysteria is probably always gradual, although it is very common for the individual symptoms to cease suddenly, especially under some profound emotion. The same agent which disturbed, in a given direction, the unstable balance of the nervous system, may readjust the equilibrium, but without rendering it more stable than before; and such influences are never permanent in their effects. Nor are they always efficient even in their limited degree. It has been said that an hysterical paralytic will always run out of a burning house, but Weir Mitchell has mentioned an instance in which, under such circumstances, the patient only fell down helpless, and another instance of this has come under my own notice. A sufficient motive for sustained, unselfish exertion is much more effectual, and many an hysterical girl has recovered health under the necessity of rising from her couch to exchange the part of invalid for that of nurse.

PATHOLOGY.—Many points of the pathology of hysteria have been alluded to in the account of the characters and causes of the disease, and our knowledge of the subject is so scanty that little more remains to be said. The teaching of anatomy is purely negative. The changes which, in rare cases, have been found on post-mortem examination, have clearly been either accidentally associated, or have merely served to evoke the condition which in other cases exists as an independent malady. The only disturbance common to all forms of the affection is that of certain cerebral functions, and we are not justified in looking beyond these for the primary derangement. It is clear that whatever influence disorders of other organs (as those of the uterus) may exert, they merely excite the manifestation of a disease already existing.

Hysteria is probably the most perfect type of a functional malady. It not only consists in, but arises by, a functional disturbance, a loss changes in the finer nutrition of the nerve-elements may underlie and result from it; but they are not, even in extreme degree, recognisable by our present methods of observation. To this one exception must be made; spinal sclerosis may develop from contracture—an almost unique example of structural change from functional disturbance.

It is scarcely worth while to discuss the pathology of the individual symptoms of hysteria. The little that is known has been mentioned in their description, and the application to them of the general principles just enunciated would be merely to repeat these principles in more special terms.

Diagnosis.—The general condition of hysteria is usually recognised without difficulty. The varied and varying discomforts of which the patients complain, and for which no organic cause can be discovered, the frequent globus, and the mental state are at once readily recognised. The special symptoms, when severe, present greater difficulties, and occasionally give rise to perplexing diagnostic problems. Not less difficulty is presented by the cases in which symptoms of hysteria accompany and complicate other diseases. Slight organic disease may be overlooked in the presence of hysteria, while the opposite error, that of mistaking hysteria for organic disease, is more frequent in the severer special manifestations of the functional malady.

A very important element in the diagnosis of the special forms of disease is furnished by their conditions of origin. The patient is of the sex and age in which the tendency to hysteria is strongest, or, if a man, presents those nervous and somewhat effeminate characteristics which usually accompany the disposition to the disease in the male. These considerations receive additional weight from the presence of the slighter and more constant indications of hysteria which have been already alluded to. But it cannot be too strongly insisted upon that this element in diagnosis, although of great value, is second in absolute importance. The first and most important consideration is the absence of any unequivocal symptom of organic disease. Women who suffer from other diseases of the nervous system are frequently also the subjects of hysteria, and it is clear that, in the presence of distinct symptoms of organic disease, the evidence of hysteria is of no significance as regards the nature of the malady, and only becomes significant when the absence of evidence of such other disease has been ascertained. The first step in diagnosis is therefore to search for any symptoms which indicate organic affections, and this involves a knowledge of the diagnostic symptoms of almost every other disease of the nervous system, since there is scarcely one which may not be simulated by this Protean malady.

In the absence of such unequivocal evidence of organic disease, the presence of hysteria, or of the conditions favorable to its existence, becomes of important significance. It does not, however, in itself constitute evidence that the symptoms are due to this cause; the aphonia and hysterical hemiplegia. A laryngoscopic examination showed that one vocal cord was completely paralysed, the other moving freely. Laryngeal paralysis due to hysteria is double; unilateral paralysis is always of organic origin, and the case proved to be one of syphilitic disease at the surface of the medulla oblongata.

It is unnecessary to consider in detail the diagnosis of each of the forms of hysterical disorder, since their leading and characteristic symptoms have been already described, and the diagnosis rests, in each case, on the detection of these characters, and on the general principles just enunciated. The chief differences from the several diseases have, moreover, been considered in the description of the latter. The distinction of the severe convulsive attacks from those of epilepsy is described in the section on the diagnosis of the latter disease.

PROGNOSIS.—In no disease does the prognosis, as regards life and recovery, present greater disparity. The danger to life from hysteria, even in its most severe forms, is extremely small. Very rarely exhaustion, consequent on vomiting, &c., terminates in death, and still more rarely severe laryngeal spasm (or more probably paralysis) has led to a fatal result. Other affections, however severe, are practically devoid of danger.

The prognosis as regards recovery varies with the circumstances of the individual case, according to the severity of the disease, and to the efficiency with which appropriate treatment can be adopted. As a rule, the special symptoms of hysteria can be removed, but the morbid state of the nervous system, of which they are the manifestations, persists in greater or less degree, so that it has been said that complete recovery is as rare as is death from the disease (Jolly). This is an exaggeration, but it is certainly true that medical skill can never, by itself, secure recovery, although, if the patient's conditions of existence are favorable, if a life of active work and satisfying aims can be substituted for the purposeless years which commonly succeed puberty, it is by no means rare for all traces of the hysterical temperament to disappear, and a stability of character to be developed, which can endure even severe trials without giving way.

Of individual groups of symptoms, some are more readily influenced by treatment than others. Motor paralysis and the slighter forms of convulsion are those most easily removed. Hysterical anæsthesia is more obstinate, and usually persists long after any accompanying loss of motor power has passed away. The local forms of spasm, both the common contracture and the rare clonic spasm, are much more enduring, and so also are hysterical vomiting, local pains, and tenderness, especially of the spine, and the severe forms of hysteroid convulsion, which are sometimes even more difficult to influence than epilepsy itself. It should be noted that the hysterical fits in which delirium is the most prominent feature, involve some danger of insanity. I have more than once known such patients to become insane. In all conditions the general law is true that the prognosis is favorable in proportion as definite causes can be traced and removed, especially deterioration in the general health, of remediable character.

TREATMENT.—The predisposing causes of hysteria are to some degree avoidable. The psychical condition from which it springs, and in which it largely consists, may be in part prevented by careful training, especially during the transition from childhood to youth and womanhood. The characters of the training needed are suggested by the description of the causes of the disease already given.

The treatment of the developed malady is partly that of the special symptoms, partly that of the underlying condition which causes them. The latter is infinitely the more important; the removal of special symptoms may do nothing for the real cure of the patient, and at best is but a small step in that direction. Nevertheless their treatment, useless alone, is a valuable adjunct to the removal of their cause.

The causes of hysteria are partly moral, partly physical; and treatment, to be effectual, must correspond. The success of the measures adopted will largely depend on a careful study of each case, and an accurate recognition of the relative amount of physical and mental disorder. The first element in treatment is the removal of whatever defect in the general health or local disorder of function can be discovered. In very severe cases, and especially after tonics and change have been tried without success, a method devised by Weir Mitchell, and extensively carried out in this country by Playfair, will often succeed. It consists in keeping the patient absolutely at rest in bed, and obtaining the tonic influence of exercise by daily massage and electricity—skilled rubbing and kneading the muscles, and putting them in action by faradism. At the same time abundant food is given in an easily digested form. By this method the wearying effects of fatigue are avoided, and patients often gain flesh and colour rapidly. It must be remembered, however, that the system is only needed in severe cases, and that the skilled rubbing and electricity are essential. Without these, rest in bed will probably convert the patient into a helpless invalid. This method has, however, another use; it affords an effective vehicle for moral treatment. For this purpose the patient is isolated during the course, except from the necessary attendant. The isolation is not only of great influence in itself, but affords an opportunity for influencing the mind, and to this the unquestionable success of the treatment is largely due.*

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* The details of the treatment are described in Weir Mitchell's books, 'Fat and Blood,' and 'Nervous Diseases in Women,' and by Playfair, 'The Treatment of Nerve Prostration and Hysteria,' 1882. Twenty years ago Russell Reynolds ("On Paralysis and other Disorders of Motion dependent on Idea," 'Brit. Med. Journ.,' Nov. 6th, 1869, p. 483) pointed out the value of massage in the treatment of these palsies.

Of organic derangements requiring special treatment, those of the digestive organs are the most frequent. They need special adaptation of the tonic treatment and careful diet. Constipation is often troublesome, and may task to the utmost the resources of the physician and the pharmacopœia. It is of the greatest importance to secure daily action of the bowels, for which moderate doses of aperients will often suffice, when very large doses are ineffectual to overcome the accumulations of a longer period. In obstinate cases, in which enemata have to be employed, it is best to combine these with aperients, and the injection should be given at the time at which the medicine ought to act; the two influences, thus united, will often be effectual when each, separately, is powerless.

The ovarian tenderness and pain, as a rule, need no local treatment; they are neuralgic in nature, depending on the general condition of the nervous system, and pass away with the latter. The treatment of uterine derangements needs much judgment and caution. It has been already stated that uterine conditions which cause no obtrusive symptoms have rarely any influence on the disease, and they should as a rule be left alone. Both amenorrhœa and menorrhagia disappear when the general health is improved. If there are symptoms that suggest positive uterine disturbance, such as sacral pain, "bearing down," or great menstrual discomfort, the question of local treatment has to be entertained. If the patient is a married woman it is well that, at the outset, the actual condition should be ascertained by a vaginal examination, and any distinct derangement put right; it being remembered, however, that mere uterine tenderness is often simply a neurotic hyperæsthesia, and is aggravated instead of being relieved by local treatment, and that even uterine displacements are not always either morbid or remediable. If the patient is unmarried, local treatment, and even a single vaginal examination, is often prolific of indirect evil, and it is better, even if the local symptoms are troublesome, to see, first, the effect upon them of the improvement of the general health and nerve-state, and only when this fails should the state of the uterus be ascertained. It is extremely doubtful whether any local condition, for the discovery or treatment of which the speculum is necessary, has, in the virgin, any influence on the origin or course of the symptoms of hysteria.

The success that has followed the removal of both ovaries in very severe and long-continued cases has been before mentioned; it may have been due much more to the indirect than to the direct effects of the operation. Whether or not this is justifiable in any case is a grave and open question. It must be remembered that there is no degree of the disease that precludes the hope of great amelioration by other means, and that the operation, while it may not lessen the malady, is certain to have its own serious consequences.

The moral treatment of hysteria, as it is the most important, is also the most difficult; needing alike insight, ingenuity, and motive force, irksome measures are sometimes necessary, adopted of course ostensibly for medical and not for moral purposes, and it is in these cases that the Weir-Mitchell system is occasionally most successful. The increase or relaxation of the strict treatment affords an effective means of punishment or reward, by which the will may be stimulated and moral inactivity dispelled. In all cases each successful effort must be commended, and the patient made to realise the progress that has been effected, and the promise of recovery which it supplies. Care must, however, be taken not to push the patient on too rapidly. When once the will has been won over, the patient is apt to try too much, and recovery from a discouraging relapse may be far more difficult than was the original advance. Power has not only to be evoked but consolidated, and the real progress is often less than that which is apparent.*

In many cases also the art of control over mind and muscle has been lost, and the will has to be re-educated by slow and gradual steps. The patient must be made to realise that deliberate endeavour, fruitless at first, will be effectual at last. A direct effort to control the onset of a hysteroid fit may at first be unsuccessful, but if the patient can learn by some expedient, such as a sudden exertion or a sluice with cold water, to keep off the paroxysm, she will gradually need less and less the adventitious help, and be able to control the fit by a mere effort of the will. So with paralysis; a deliberate and well-intended effort to walk may fail, but by learning to stand, first with support, then alone, and then to walk with help, the will slowly regains its lost power. All progress must be gradual to be enduring; haste often causes a troublesome and disheartening relapse.

It is perhaps not superfluous to caution the physician against increasing the morbid state by any proceeding of his own. Attention is a potent intensifier of the symptoms of hysteria, and by too frequent examination and demonstration, certain phenomena, such as anæsthesia, may be kept up, which would disappear if disregarded. Especially is this caution necessary regarding the induction of the hypnotic state and of convulsive phenomena, which is always harmful, and sometimes perpetuates the malady.

Apart from the tonics already mentioned, certain drugs have, for many centuries, been held in repute as powerful "antihysterical" remedies. They are for the most part substances of nauseous taste (musk, asafœtida, valerian, &c.), and their early use was associated with the theory that, by their offensive character, they drove the errant uterus back to its place. Although by some recent writers their influence for good is discredited, they are still largely employed. Most of them undoubtedly have a stimulant action on the nervous system, which is imperfectly recognised by their designation as "antispasmodics." Their flavour is usually (though not always) most perpetuated by repeated medical investigation. If its degree and persistence render local treatment desirable, faradisation with a wire brush, or sparks from a friction machine, are the most effectual. Amaurosis may be treated by stimulation of the retina by a feeble voltaic current, slowly interrupted. Loss of hearing and of taste need no special treatment.

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* The various points in management are admirably discussed by Weir Mitchell (loc. cit.).

Hysterical aphonia sometimes yields at once to exhortation or to the introduction of the laryngoscope. It can usually be removed by faradism applied to the exterior of the larynx, the patient being made to utter a sound during the operation. If this fails, sparks from a static machine may succeed. In obstinate cases, faradism to the interior of the larynx or a circular blister round the throat, is usually effectual, but I have never found the former necessary. Aphonia is very prone to relapse unless its removal is combined with radical treatment. In the defective co-ordination of the respiratory and laryngeal muscles, Weir Mitchell has found the most effectual plan is to insist on all utterances being preceded by a deep inspiration.

For paralysis of the limbs, no single remedy is so effectual as faradism. The movement of the muscles, and the pain produced, have often a profound effect. In slight cases the full power of movement may be restored by a single application. More often its use has to be repeated, and the effect is gradual, each application being followed by a slight increase in power. It is important that the patient should expect the gain in power which will follow its use. In severe cases, the calls to increased effort must be very gradual and systematic, care being taken that no attempt is allowed to fail entirely, or is prolonged to fatigue. In severe inco-ordination, the power of steady movement has to be carefully and gradually trained; if necessary, first in bed and then on "all fours," the erect position being gradually assumed. Great assistance is often derived, in both ataxy and weakness, from a steady frame on wheels, or from sticks or crutches which have a concave handle for the grasp and a large flat base, eight inches by two, covered with india rubber, as suggested by Weir Mitchell.

Contracture of limbs can sometimes be removed by an unfamiliar agent which produces a strong sensory and moral impression, such as faradism, or static electricity, or a circular blister around the limb. If these fail, more gentle and continuous measures must be adopted. Violent physical force is seldom successful, and not rarely does harm, failing to relieve the symptoms, and inducing other nerve troubles. Occasionally, contracture may be removed by chloroform, and the relaxed limb fixed in some other posture before the patient recovers consciousness. More permanently successful, however, is rubbing (which rarely fails, for a time, to lessen the contracture), combined with gentle passive movement; the position of the limb may be slowly changed by means of splints. The voltaic current is rarely of service. When the contraction is lessened, systematic voluntary ovement should be added. Many forms are extremely obstinate, d their treatment requires great patience and perseverance. Con ction of the muscles of the jaw is an exception, and generally dis ears spontaneously if disregarded.

hantom tumours in the abdomen are best left alone. They cause little inconvenience, and are often perpetuated by local treatment. Faradism to the muscles which are relaxed will lessen the prominence during the application, and this, somtimes combined with rubbing, may be employed if any local treatment is thought desirable.

The tremor-like forms of clonic spasm are merely accessory to the weakness and contracture, and disappear with these. More violent local spasms are often removed by a circular blister around the limb, but they are occasionally extremely obstinate, and resist all symptomatic treatment, yielding only to the radical amelioration of the hysteroid state. This is true also of rhythmical spasm, which, however, as a rule, is more amenable to treatment, and is sometimes arrested by hypodermic injection of morphia, atropine, or arsenic. The occasional use of morphia for such a symptom entails less danger than its employment for the relief of pain.

The visceral symptoms of hysteria also give great trouble. In anorexia and refusal of food, the patient must be compelled to take liquid or pulpy food, and it is often best for the nurse to feed the patient. If even this is refused, the nasal tube may be employed, or rectal injections; these operations are often interfered with by the occurrence of hysteroid convulsions, which usually prevent the employment of the stomach-pump. The nasal tube should not be deferred too long; its moral effect is very great, and may save much further trouble. By means of this, or the œsophageal tube, food may be given prepared as described below.*

Hysterical vomiting is often most troublesome, both on account of its obstinacy and of the impaired nutrition to which it may give rise. It is best treated by absolute rest, liquid food, and the spare feeding which rest renders adequate. The rejection of food should be met by judicious disgust, and a more liberal diet may be promised on its cessation. Severe vomiting is one of the symptoms for the treatment of which seclusion is most useful. When other means fail, forced feeding by the stomach-pump has been successful. The avoidance of the act of swallowing seems to have something to do with the retention of food, since when injected food has for some time been retained, that taken in the ordinary way may still be vomited (Dujardin-Beaumetz).

* Debove, who has largely employed this forced feeding, recommends that the daily quantity should be 5 pints of milk, 1½ pounds of meat, 12 eggs, and the flour of cooked and dried lentils. The raw meat is minced, warmed, pressed, placed in a stove and dried thoroughly, and reduced in a mortar to an extremely fine powder. The lentils should also be cooked and then pounded. The whole is mixed with the milk and with the beaten-up eggs, and given in four or five injections into the stomach ('Gaz. méd. de Paris,' 1882, p. 206).

The vaso-motor symptoms of hysteria rarely yield to direct treatment. Occasionally digitalis lessens the palpitation, and the flushings and cardiac discomfort are relieved by belladonna; but in spite of the apparent independence of these troublesome symptoms, the radical treatment of the disease is usually the only means of affording more than temporary relief.

The treatment of the cases which present hysteroid convulsions must vary according to the nature of the seizures. It has been mentioned that the attacks, of which such convulsions are the conspicuous feature, may be really compound, consisting of a true epileptic fit and of hysteroid convulsion as a post-epileptic phenomenon. In such cases, the treatment has to be that for epilepsy, and bromides do most good. Where, however, there is no real epileptic element, and the attacks are purely hysteroid, bromides usually fail, and other agents ometimes succeed. Moral treatment has a great influence on many these cases. The attacks often cease at once when the patient is mitted to a hospital. If they do not, she should be urged to deavour to control them. The attempt will often be aided by the ect of drugs in rendering the attacks less severe, and the patient ould be impressed with the fact that attempts at control, which may ve failed before, will, thus aided, be more successful. When direct trol fails, various expedients may afford help, as cold water, olling-salts, &c.

Drugs exert more influence on the convulsions than on many of other symptoms of hysteria. Those that are most useful are valerianate of zinc, iron, morphia, and turpentine. Iron appears do good by a special action on the nerve-centres, apart from its atinic effect, and is especially useful in the hysteroid attacks h occur in boys. Turpentine may be given in ten-minim doses, ually increased until slight symptoms of strangury are produced, it must be omitted for a few days. It is perhaps more useful any other single remedy.

e onset of slight hysterical seizures, emotional in character, may be averted by an "antispasmodic" draught of ether, lavender, and sometimes by the inhalation of nitrite of amyl. The deed attack may also be usually arrested, although the means most al for this are not the same in all cases. A douche of cold on the head often succeeds, but it sometimes needs to be very s; the first jugful may only increase the violence of the convuland a second may arrest it. When so much water is needed, it umsy remedy. A few teaspoonfuls poured into the mouth, or, is closed, into the nostrils, are often at once effectual, apparby stimulating the respiratory centre. The same result may e effected by Dr. Hare's plan of closing the mouth and nose wel for fifteen or twenty seconds. Sternutatories usually fail. method of cutting short an attack is a painful impression. n (or other hysterogenic) tenderness is known to exist, firm medicine is small. The phenomena are chiefly important from their correspondence with the similar states that arise spontaneously. Hence a very brief outline of the most important facts may be given.

INDUCED HYPNOTISM.

Three different conditions may be produced in the hysterical, and have been thus summarised by Charcot:*

1. A *cataleptic* condition may arise under the influence of some loud unexpected sound or dazzling light, or persistent fixation of an object by the eyes. It may also occur as a sequel to the second form, the state of lethargy, if the eyelids of the lethargic patient are raised in a well-lighted room. The cataleptic condition is characterised by rigid fixation of the limbs, with open eyes, a stiff look, and slow breathing. In spite of the fixation of the limbs, they can be moved passively with great readiness, and remain in whatever posture they are placed in, without apparent fatigue. Very little evidence of myotatic irritability can be elicited. The skin is completely anæsthetic, but the organs of special sense retain their sensibility, at least in part, and through their means the subject can be induced to perform automatic actions of a more or less complicated character, by imitation, or by the suggestions of description, but when left to herself relapses into the state of statuesque rigidity.

2. The *lethargic* condition. A person in the cataleptic state just described may pass into the lethargic condition on the occurrence of a deep inspiration, or the transition may be induced by closing both eyes, or by darkening the room. The condition may be primarily induced by prolonged fixation of the eyes. In this state the eyes are closed or half closed, and the globes deviate upwards and inwards. There is complete relaxation of the muscles, and the limbs fall when raised. Myotatic irritability is greatly augmented, and there is an extraordinary increase in the mechanical excitability of the nerves and muscles, so that contraction follows not only a tap on the muscles but pressure on the nerves. A pointed object pressed on the "motor point" of a muscle will put it in action just as does faradism. In the limbs and trunk the contractions thus produced continue; they may even persist after the patient has emerged from the lethargy, and can only be removed by stimulation of the antagonists. In the face, the contraction ceases when the pressure is discontinued. There is complete anæsthesia, not only in the skin but also in the organs of special sense. If the eyelids are raised in a lighted room, the lethargy passes into the first condition, that of catalepsy, and if only one eyelid is raised, the change occurs only on the corresponding side, so that the two conditions co-exist and can be contrasted.

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* 'Progrès méd.,' 1882, p. 124; and 'Leçons Mal. Sys. Nerv.'

remain in any position in which they may be placed. The resistance to passive movement is peculiar; it is as if the limbs were made of wax, and hence the condition has been termed *flexibilitas cerea*. The rigidity commonly yields slowly to gravitation. The countenance is usually expressionless. The respiratory movements and heart's action are weakened. Substances placed in the back of the mouth are swallowed, but slowly. The state of sensibility varies; in profound conditions of catalepsy it is lost to touch, pain, and electricity, and no reflex movements can be induced even by touching the conjunctiva. In other cases partial sensibility remains, and reflex phenomena may be excited. In rare instances paroxysmal hyperæsthesia is present. Consciousness is frequently lost, but may remain more or less obscured, rarely intact. The temperature is commonly lowered. The attack may last a few minutes or several hours. Recovery is gradual or sudden; it is common for the patient at first to be unable to speak. Sometimes a strange periodicity may be observed in the occurrence of the paroxysms. In the intervals between the attacks, headache, giddiness, or hysterical manifestations may be present, or the patient may feel and seem perfectly well.

PATHOLOGY.—In the passive movement of a limb, the muscles have their course lengthened or shortened, by the approximation or recession of their points of attachment, yet they remain in a state of tonic contraction uniformly adapted to their shortening or elongation. This must be the result of adjusted activity of the motor cells of the spinal cord, determined by the afferent impressions from the muscle-nerves, the result of increased or lessened tension; we have seen (vol. i) that tension is one of the modes in which they are stimulated. If the adjusted activity of the cells and tonic contraction of the muscles are abnormally great, although varied in normal proportion, the state of the limb must be that which exists in catalepsy. We cannot be wrong in assuming this mechanism. But the spinal cells are influenced by those of the cerebral cortex, which are apparently also regulated by afferent impulses from the muscles (see vol. i, p. 202), and it is probable that the over-action arises here, and that the state of the spinal mechanism is secondary. This is probable, first because no simple excess of the action of the spinal centres, known to be such, gives rise to the phenomenon; and secondly, because the state can be produced by influences acting on the cortex, and disturbing its higher functions. The lower cortical centres seem to be controlled by the higher, and the control is evidently deficient in hysteria, in which catalepsy especially occurs. A deficient control of the motor centres of the cortex, permitting their over-action, regulated by the afferent impulses, and repeated in the spinal cord, is the best theory we can at present frame of the process of catalepsy.

The DIAGNOSIS of the condition presents no difficulty. Many cases of simple trance have been included under the term "catalepsy," but the immediate excitant, or an attack may follow an hysterical convulsion. Weir Mitchell has described a case in which conversation on any unpleasant subject would bring on an attack. In one celebrated case, that of Colonel Townsend, the state could be induced voluntarily, and in one attack so induced he died. Voluntary induction is not uncommon in the East. The production of the state in hysterical subjects has been already described.

A similar condition is sometimes observed in the insane, especially in the subjects of chronic delusional insanity. Sleep, during which liquids are swallowed, may last for four or six weeks. Several such attacks may be separated by intervals of a few days or a fortnight. They occur without obvious exciting cause.*

SYMPTOMS.—The onset of the common form of trance is usually sudden. In one case that came under my notice, a girl went into a room by herself, and was found, shortly afterwards, in a state of trance-sleep that lasted forty-eight hours. Another case, recorded by Madden, came on in a similar manner, and lasted for a fortnight. The onset has been occasionally attended by a sensation resembling the globus hystericus. In the cases after typhoid, the delirium of the fever has passed into a condition of comatose sleep that lasted for several weeks; this sequel may be connected with the marked mental weakness or loss of speech that sometimes succeeds this disease. After influenza, a patient has suddenly passed into such trance-sleep, and has wakened spontaneously after a few hours or days.

During the trance state the face is usually pale. The limbs are relaxed, as a rule, throughout the attack, but sometimes there has been brief initial stiffness, or occasional transient cataleptic rigidity; tonic spasm and distinct hysteroid convulsions have occurred from time to time in some cases. The eyelids are closed, and attempts to open them are resisted. The eyeballs are usually directed upwards and to one side; in hysterical trance they sometimes converge. The pupils are moderately dilated or contracted, and almost always act to light. Reflex action in the limbs has been lost in deep trance, increased in slight cases, and cutaneous stimulation sometimes induces muscular contracture. The reflex action from the conjunctiva and nose may be lost, and pressure on the ovarian region, if this was tender, may have no effect. In profound trance the mental functions seem to be in complete abeyance, but in slighter degrees the patient may be aware of all that passes, although unable to make even the slightest manifestation of consciousness. The special senses may even be unnaturally acute. Occasionally there is evidence of spontaneous mental action, analogous to dreaming, manifested by exclamations, and even by movements; in rare cases there is the "obedient automatism" so conspicuous in the induced trance, in which hallucinations can be generated, and actions excited, by suggestions made to the patient.

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* See Szezypiorski, 'Ann. méd.-psych.,' Nov., 1891.

local cerebral lesion or a toxic cause. Other diagnostic symptoms are the pallor and vascular depression, the occurrence of convulsive phenomena of hysteroid type, and the previous existence of other manifestations of hysteria. These symptoms sufficiently distinguish trance-sleep from apoplexy, for which, at the onset, it is sometimes mistaken. The distinction from catalepsy rests on the muscular relaxation.

In cases of "death-trance," in which no sign of vitality can be recognised, the presence of life may be ascertained (1) by the absence of any sign of decomposition; (2) by the normal appearance of the fundus oculi as seen with the ophthalmoscope; (3) by the persistence of the excitability of the muscles to electricity. This excitability disappears in three hours after actual death. In a case observed by Rosenthal, thirty hours after supposed death the muscles were still excitable, and in forty-four hours the patient awoke.

PROGNOSIS.—In most cases of lethargy the ultimate prognosis is good. The slighter the degree of the trance the shorter is likely to be its duration. The prognosis is grave only when the lethargy has been preceded by a state of great physical depression, especially when the condition has succeeded an acute disease.

TREATMENT.—The treatment has to be directed to two ends; the maintenance of life, and the arrest of the trance. Advantage must be taken of any intervals of semi-consciousness to give nourishment in a concentrated form. If swallowing is continuously impossible, food must be given by the nasal tube, or by enemata. Warmth should be applied to the extremities, and care taken to prevent bedsores. In severe cases, every attempt at arrest is often fruitless. In cases of moderate severity stimulation of the skin is effective in rousing the patient. The most powerful cutaneous excitant is strong faradism. In one case, which had lasted for thirty-six hours, I found that the application of strong faradism to the arm quickly cut short the trance. In another case, which lasted for several months, this treatment had for a long time no influence; afterwards the patient could be partially roused for a short time by faradism, and by repeating the application at the same hour every day, a tendency to periodical waking was established, the remissions became longer and more complete, and the attack was ultimately brought to an end. Nervine stimulants, such as ether and valerian, may be given by the bowel, or sulphuric ether may be injected subcutaneously. Alcohol must be given with caution and in small quantities; enemata of strong coffee are often more useful. It is probable that nitro-glycerine, or the inhalation of nitrite of amyl, would have considerable influence. Transfusion of blood has been proposed, and would be justified in cases following exhausting disease when death is threatened. The recurrence of attacks must be prevented by the improvement of health, physical and moral.

African Lethargy.—A curious malady, presenting similar somnolence but of very different nature, is the "sleeping sickness" of the West Coast of Africa, met with chiefly in the Congo and Sierra Leone regions. It is said to prevail less at the coast than inland, and to be especially severe in the valley of the Congo. It affects exclusively negroes (by whom it is much dreaded), and occurs in both sexes and at all ages, but is most frequent in males between twelve and twenty. Except that depressing emotions seem to predispose to it, the proximate causes are entirely unknown. Europeans living in the same localities are exempt. Swelling of the cervical glands sometimes occurs at the onset, and they are excised by the native doctors as a remedial measure; but the condition is not invariable, and its influence is doubtful. The general health at first is good. There is a gradually increasing tendency to somnolence, and the patient will fall asleep at various and unusual times, at his work or over his meals. A tendency to persistent drooping of the upper lids sometimes occurs at the onset. At first he can be roused, and if treated by cutaneous stimulation and purging, the symptoms may be removed for a little time, but they soon recur and increase in spite of treatment; the periods of sleep grow longer and more frequent until the patient is always asleep, is reduced to a merely "vegetative" existence, and ultimately cannot be induced to take food. He gradually emaciates, and dies at the end of three, six, or twelve months from the onset of the symptoms. Just before death the disposition for sleep often ceases. The disease is extremely fatal. Guèrin met with 148 cases, all of which died. The observations of Gore and others place the mortality somewhat lower—at about 80 per cent. Post-mortem examination has revealed only hyperæmia of the arachnoid, slight signs of chronic meningitis, but no considerable excess of fluid within the ventricles or outside the brain. The cerebral substance is usually pale. *Filaria sanguinis* has once been found in the blood,* but apparently only as a coincidence. In some cases with organic brain disease, it would seem as if the symptoms had acquired a special character from the cause of the lethargy, whatever it is. Poisoning by a fungus growing on cereals has been suggested, but the malady, once established, progresses steadily in spite of change of residence and of diet. No treatment appears to influence the symptoms. Only one observer (McCarthy) has seen good from excision of the cervical glands. This mysterious affection—mysterious in more respects than are recognised by those who have theorised about it—clearly needs more systematic investigation than it has yet received.†

It is clear, moreover, that a tendency to somnolence, increasing gradually to lethargy and coma, is the expression of morbid blood-states more often in tropical countries than in temperate regions. Such cases have been observed in South America, attended with pyrexia, with only trifling secondary changes in the brain, but with the enlargement of the spleen that shows a morbid state of the blood. The symptoms of these cases differ, in essential particulars, from the African malady.

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* S. Mackenzie, 'Lancet,' 1890.

† The most recent account of the disease is by Junker v. Langegg, 'Wien. med. Wochenschr,' 1891.

HYPOCHONDRIASIS.

Hypochondriasis is a morbid state of the nervous system in which there is mental depression due to erroneous ideas of such bodily ailments as might conceivably be present. This limitation is necessary to distinguish the condition from those forms of actual insanity in which there is a delusion of the existence of some impossible ailment —impossible either in its nature or else by reason of its incompatibility with life. A patient, for instance, who thinks that his spinal cord has become detached from the brain, or who believes that his throat is hermetically closed, is insane, not hypochondriacal.* The term is, moreover, almost exclusively applied to the condition above defined when this is met with in men. A similar state in females is regarded as forming part of "hysteria." The name has come down to us from the times of Hippocrates and Galen, and, like "melancholy," had its origin in the idea that the conscious life was largely influenced by "vapours," &c., produced in the abdominal organs. It was not until the early part of the present century that the disease was generally admitted to be one of the brain.†

Causes.—The affection consists in a particular disposition of the brain, which leads to habitual anxiety without adequate cause, and to the concentration of this anxiety on the individual's own health. This morbid tendency is often a matter of individual temperament, which may be the result of neurotic inheritance; the most severe forms occur in persons whose family presents a tendency to insanity. In other cases there is no morbid heredity, but the state is acquired through persistent ill-health, which sets up the morbid mental habit of self-attention and concern. These two causal varieties may be termed the "acquired" and the "temperamental" forms. The age at which hypochondriasis comes on varies according to its causes. It is scarcely known in childhood, but when due to inherited temperament it often becomes established quite early in adult life, and sometimes is even distinct in youth. It may, however, commence at any period, and the acquired form often commences in or after middle age.

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* It is unfortunate that alienists sometimes apply the term to the cases of insanity with somatic delusions; this is contrary to its customary use, and productive of confusion.

† An interesting sketch of the history of hypochondriasis is given in Gull and Anstie's article on the disease in Reynolds' 'System of Medicine,' vol. ii.

Hypochondriasis seldom develops without some exciting cause in the shape of a distinct bodily ailment, although this is often very trifling in degree. It suffices, however, to give rise to unpleasant sensations, which excite attention and give rise to concern. Dyspeptic troubles are the most common excitants; others are palpitation of the heart, and weakness of the nervous system; but there is hardly any part of the body in which functional disturbance may not be the starting-point of the malady. When the attention has once become fixed on a bodily sensation, and this has become the basis of a fancied malady, other feelings elsewhere are soon perceived; corresponding ailments are imagined, and any trifling disorder is magnified into a serious disease.

SYMPTOMS.—In hypochondriasis there are three chief elements: excessive anxiety regarding the personal health; undue attention to any indications of derangement, especially to the various sensations emanating from the organs of the body; and lastly, as the consequence of these, there is some false idea that disease is present which does not exist. Occasionally the last is absent; the patient may know and realise that he is free from any serious malady, but he is rendered miserable by the continuous discomfort he experiences.

This discomfort, as already stated, often has its origin in some actual disorder, but it is always vastly increased by the habitual attention paid to the sensory impressions proceeding from the organ deranged, or from other parts when there is no local disorder. The influence of attention has been already mentioned in the account of "cephalic sensations" (p. 863), which often form part of the symptoms of hypochondriasis, and the statements there made are applicable to all the various sensory impressions from other organs that disturb the hypochondriac's life. It is this continuous attention that perpetuates the morbid state, and renders its treatment so difficult. Not only are sensations attended to until consciousness is continually under their influence, but the patient is always searching for indications of the ill-health he dreads. He scrutinises his tongue before breakfast and his evacuations after breakfast, and notes, during the day, the influence of each meal on his abdominal feelings, of exertion on his pulse, and of mental work upon his head. It is easy for the hypochondriac thus to collect each day a series of "symptoms" which he ponders over, endeavours to interpret, and sometimes records, in minute detail, in a diary. As an instance of this morbid self-observation and attentive regard to every real or fancied sensation, I may quote *verbatim* the description given to me by one of these unfortunate sufferers, in whose physical condition no other flaw could be found grave. Occasionally, a temperamental hypochondriac may be met with who describes life as shorn of all pleasure by his sufferings, and asserts that he has suicidal impulses; but if the latter really exist, there is generally more than pure hypochondriasis.

To describe in further detail the symptoms of these cases would be merely to multiply particular illustrations of the general facts above stated. There is hardly a malady that is not at times the subject of the morbid fancy. The general facts are true of all; it is only the applications that vary. One class of cases, however, commonly included in this category, deserves special mention, the cases of sexual hypochondriasis. Most of those who wrongly imagine that they have some ailment of the sexual organs do not really suffer from hypochondriasis. This erroneous belief is justified because it is founded on evidence which is adequate to cause the belief in those who have no means of estimating its value. The idea of spermatorrhœa, however, sometimes does concentrate the attention on sensations from those parts, and may set up true hypochondriasis, by the tendency to attribute to the supposed disease any discomfort that may be experienced. But there is no such disease as spermatorrhœa. The slow escape of the continuous secretion from the testicles is a normal and necessary event; neither it, nor the process of secretion, has the slightest influence on the nervous system.

DIAGNOSIS.—The diagnosis of hypochondriasis depends, in the first place, on the exclusion of the disease from which the patient imagines that he is suffering. This of course involves an accurate knowledge of the symptoms of each malady, and it is necessary to exclude real disease before the obvious mental state can be allowed to influence the diagnosis. The danger that a mistake of this character should be made depends on two circumstances. The first is the neglect of the consideration just mentioned; the manifest temperament leads to a hasty diagnosis, and deters from thorough examination. It is obvious that hypochondriasis does not exclude organic disease, and that hypochondriacs not only may, but at some time must, suffer from such disease. Secondly, error is sometimes due to the fact that the patient suffers from some uncommon malady, the symptoms of which are unfamiliar or unexpected. Thus a man with very distinct symptoms of diphtheritic paralysis had been told that these were all due to nervousness and fancy, because the patient was a member of the medical profession, the diphtheria had been doubtful, and it had been acquired in an unusual way—by inoculation.

The other diagnostic question is the distinction of hypochondriasis from insanity. It is in the permanent hypochondriac, with a family history of insanity, that this difficulty arises. The distinction depends chiefly on the reasonableness of the false ideas. A patient who has unpleasant sensations in the abdomen, and believes that he is suffering from cancer, in spite of the assurance that he is not cannot on this refusal, if the grounds for it are made clear, will do more real good to the sufferer than can be achieved by any other means.

NERVOUS WEAKNESS; NEURASTHENIA.

It has become fashionable to apply the term "Neurasthenia"* to conditions of weakness of the nervous system. The use of the word has brought with it a tendency to regard the condition thus denoted as a definite disease. Books have been written about it, and it has been divided into numerous classes according to the character of the symptoms that are present, and the causes that give rise to it. It is convenient to be able to designate the condition by one word instead of two, but there is no more justification for regarding neurasthenia as a definite malady, as a disease due to a definite morbid process, or even as an affection characterised by a well-marked group of symptoms, than there is for adopting a similar course with regard to "debility" among general diseases. "Neurasthenia" underlies and may cause a large proportion of the functional diseases of the nervous system, and when "neurasthenic" patients seek advice, there are, in many cases, symptoms of nerve disturbance sufficiently definite in character to bring the case into some special category. Moreover, the conception has furnished material for the exercise of an unlimited amount of intellectual ingenuity, in a *quasi*-scientific process of elaborate description and multiple classification. Like other general terms, it has a tendency to hinder investigation, and to prevent the thorough study, and the due discrimination, of the nature of maladies, essential alike for the progress of medicine and the treatment of disease.

But the term has taken possession of medical thought in every country to an extent which compels its adoption. The cause which has rendered it so effective is obvious. While the undue extension of the use of the word, conspicuous in many quarters, is to be regretted and deprecated, there are many cases for which it is a convenient designation, and to which it may be applied without other disadvantage than the indirect sanction that may seem to be given to its unwise use. These are the cases in which there are symptoms of disturbance, or of impaired function of the nervous system, more or less persistent, almost infinitely various in their features, but which have in common the negative characteristic that they do not conform to any of the types that have received definite names. To include the latter under the term is for many reasons unwise. Hypochondriacal, hysterical, and many other varieties of "neurasthenia" have been described; but if the fact that impaired nutrition and strength of the nervous system is to be made the ground for including under the term all the maladies which result from the condition, half the functional and nutritional affections of the nervous system should be described in one huge chapter under this designation. Equally unwise is it to distinguish classes according to the cause of the condition, when its manifestations present no peculiarities corresponding to the particular causes. Such varieties as "syphilitic neurasthenia," "influenzal neurasthenia," "malarial neurasthenia," have no scientific justification.

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* Invented by an American physician, Dr. Beard.

Even among the cases of nerve disturbance which cannot be placed in any recognised category, there are some that ought not to be included in this, or, indeed, in any other class of actual disease. Many persons possess a congenital peculiarity of nerve-function, for which they may seek medical advice, but which is not really morbid. Such is the persistence of the "shyness" of early life, or a tendency, life-long, to look on the darker side of things, or the vaso-motor activity which causes so many persons, all through the first half of life, to blush at the least emotion, and flush under every favorable physical influence.

When these, and the cases of a definite, long-recognised type, are excluded, those to which the terms "neurasthenia" and "neurasthenic" may be applied with convenience and without disadvantage are sufficiently numerous. In many the condition is distinctly "constitutional;" that is, the defect in the nervous system is inherent in the individual, and a similar ancestral tendency can often be traced. It dates, in some, from childhood; in others, it comes on after puberty or in early adult life, without any discoverable cause. Of the latter, a large proportion are females, who are unable to bear even the average strain of life, and break down in various ways. They may be raised to a little higher level of nervous health, but cannot be made really strong. Males sometimes suffer in the same way, but in them depressing influences can more often be traced. In both sexes the state often results from definite diseases of the nervous or general system or of other organs, which leave lasting impairment of nerve strength. Various influences, of occupation, amusement, dissipation, excess, may induce the condition; to enumerate these in detail is scarcely necessary.

The deficient strength implied in the name, persistent as it is in the cases thus designated, is always the expression of some degree of impairment of nutrition in the nerve-centres, brain or spinal cord, or both. It generally involves many parts, and is expressed by symptoms of great diversity; those of a certain class preponderate in some cases, but in many, various derangements of function alternate or co-exist. As a rule, deficient capacity for functional activity is associated with an undue readiness for slight action, which may even occur without its usual causes.

Sensory symptoms are the most common, but are confined to those of "irritative" character. Loss of sensation does not exist in simple "neurasthenia," but subjective sensations are almost constant. Fatigue may be produced with undue readiness by muscular exertion and by mental effort. Headache is common, and so also are the varied cephalic sensations described at a preceding page. Pains in the back are frequent, sometimes felt throughout the spine, sometimes chiefly in the sacral region; they are usually dull and "aching" in character. Spinal tenderness, often at more than one place, is frequent in women; walking and standing readily increase the discomfort in the back, sometimes so as almost to incapacitate the sufferer. In the limbs, slight fugitive pains are often coupled with varied sensations of tingling, numbness, and the like, which may be greater on one side than on the other. Muscæ volitantes annoy and alarm the patient, and when there is hypermetropia, the ciliary muscle is unable to maintain its usual contraction, so that "asthenopia" is added, and the use of the eyes begets uncomfortable sensations in them, superficial or deep, seldom painful, although actual pain may be felt behind the eyes or in the forehead, after their prolonged use. Auditory hyperæsthesia may cause pulsating tinnitus, or, in its central form, may render every loud sound distressing.

Muscular strength is only lessened in the severer degrees of nervous weakness, but the power of sustained exertion is generally reduced; fatigue is not only sooner felt, but is often a more unpleasant sensation than the fatigue of health, and whatever pain or discomfort to which the sufferer is liable is apt to be induced. Even talking may quickly cause a feeling of weariness and any cephalic sensations to which the patient is liable. Many of these sufferers habitually talk in a low voice, as if every sentence involved an exertion almost beyond their strength. A sense of muscular inertia and powerlessness is very frequent, especially in the earlier part of the day, when there is no real lack of strength; the effort needed for any exertion may seem beyond that which is possible.

Symptoms of motor irritation or over-action are very rare except in trifling form. The sense of stiffness in the limbs is not accompanied by any real rigidity. Some sufferers are very liable to cramp in the calves or feet. Especially common is the quivering contraction of some muscle, or part of a muscle; the orbiculares, glutei, thigh or calf muscles are frequent seats. It seems due to the intermitting, instead of continuous, release of the nerve-force which causes muscular tone, and has no other significance than a slight general impairment of nutrition and function, although it often causes grave anxiety. Restlessness is common, and sometimes an unendurable sense of "fidgets" causes movements almost choreic in their character and constancy, accompanied even by actual starts of the limbs.

When there are sensory symptoms in the legs, or undue readiness and degree of fatigue on standing and walking, the knee-jerk is often increased, perhaps from the diminished resistance in the reflex centres, due to the fall of nutrition below the normal standard. No foot-clonus can be obtained. The state of the sexual functions, in the male, depends chiefly on antecedent influences, but the mental preoccupation, engendered by the varied sensations which absorb the mind, and the sense of disability, deepened by a fear of impending calamity, often cause an apparent failure of desire and power, superficial only, and ceasing with its cause. In women, the state of the menstrual functions depends more on the general health than on the state of the nervous system, although the latter may be depressed by mental suffering, or by the apprehension of grave results of irregularity.

But as the most special, elaborate, and complex part of any system is that most prone to suffer, and exhibits in greatest degree the effects of general impairment, the functions of the brain seldom escape disorder in these cases, and often the cerebral disturbance is the most pronounced manifestation of the state. All the functions are apt to suffer; mental power is lessened, the ability to concentrate the attention fails, memory is imperfect, and the influence of the will upon it, in "recollection," is weakened in especial degree. There is as much conscious difficulty in mental as in physical exertion, and, in proportion as the sufferer shrinks from the unpleasant effort, the resistance that has to be overcome seems to increase. All cerebral processes that depend on the will quickly cause fatigue, which is expressed by varied sensations, psychical or cephalic. The subjective discomfort in the limbs and back depress the brain, on which, often, the effects of the varied sensory symptoms seem to be focussed. A large proportion of these, indeed, arise in the sensory centres of the brain, and the interaction of all its parts is such that the disturbance of one region influences the whole. The purely mental functions suffer with the rest; mental depression is conjoined with irritability, and every annoyance is magnified into a disaster. While actual giddiness is uncommon, a sense of impending giddiness sometimes causes much distress, and may even, by some indirect mechanism, induce uncertainty of movement.

Visceral symptoms, pain, discomfort of many kinds, and even definite disorders of subordinate functions are frequent, but those of the digestive system are of especial importance. They are to be recognised, as distinctly augmenting other symptoms, in about two thirds of the sufferers, and a close relation between gastric disorder and cephalic discomfort is especially conspicuous. A feeling of shortness of breath, or oppression at the chest, is often complained of. Secondary disturbance of the action of the heart is frequent, and the readiness with which the vaso-motor system responds to central and visceral derangement, is the source of many symptoms, flushing, pallor, sense of heat and cold, chills, and imaginary fever, are common complaints. Sleep is seldom good, restlessness and insomnia increase the nerve prostration, and often, when the patient does sleep, he wakes unrefreshed, weary, and languid. Starts often hinder the advent of sleep, and this, when it comes, is disturbed by dreams or interrupted by sensations of tingling in the hands, sometimes with a feeling of contraction in them when there is none, sometimes with an actual contraction such as that described at p. 704 as "nocturnal tetany."

The further development of such effects of malnutrition usually takes the form of one of the definite functional ailments already described, many of which, as neuralgia, hypochondriasis, hysteria, really consist in a high degree and elaborate development of the effects of the condition now under consideration. The description of the symptoms might be multiplied indefinitely, and yet, so various are they that no description, however lengthy, would embrace half the varied manifestations of mere nervous weakness.

PROGNOSIS.—The prospect that the condition will pass away must depend on its duration, and especially on the degree to which it is the effect of influences that can be arrested, or, on the other hand, is the manifestation of an inherent inability of the nervous system to reach or maintain a proper level of nutrition and function. Each case must be made the subject of separate study, and the causal influences must be compared with the extent to which the interaction of the symptoms tends to keep them up, and with the possibility of emancipating the sufferer from the pathogenic influences, external to and within himself. When patients have been freed from the acquired state, it is extremely prone to return on any impairment of the general health or strain upon the nervous system.

TREATMENT.—The advice that has been given regarding the treatment of hysteria, hypochondriasis, neuralgia, headache, and cephalic sensations, embraces most that can be done to remove the condition of "neurasthenia." All influences conducive to physical strength should be secured, and all causes of depression and exhaustion of the nervous system should be sought for and eradicated. Powerful causes (as sexual excess) are sometimes quite unrecognised as such by their victims. A sea voyage and mountain air afford the most potent means of restoring the strength of the nervous system, if this has been lately lost. In all cases, rest from work that is obviously depressing should be secured, and the rest made more effective by such mental and physical influence as can be secured by change of personal and local surroundings. In many, however, some mental occupation is better than perfect freedom to devote attention to the sensory discomfort. Tonics should be given of such form as is indicated by the symptoms, and drugs that lessen the instability of the sensory structures, without depressing the system, are often of service, such as small doses of Indian hemp, given with bromide, belladonna, or chloride of aluminium. The effect of nitro-glycerine, in augmenting the arterial blood-supply, and thus facilitating improvement in nutrition

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